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Table of Contents

An Address.[1]

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THIS is the first occasion on which the annual meeting of the Royal Australasian College of Surgeons has been held in Adelaide. The Council in its generosity has more than once suggested a meeting in this city. I have always demurred because it has seemed to me that to the general body of Fellows the larger centres of hospital and university work like Melbourne and Sydney make stronger appeal, but perhaps the warmth of Adelaide's welcome and the quality of what the visiting Fellows will see and hear may help to compensate for other shortcomings.

My Council has suggested that the theme of this address should be historical in nature, and deal particularly with the aims and objects of this, the youngest of the Royal Colleges of Surgeons of the British Empire. I propose first of all to delve in a desultory way into the past, for notwithstanding that surgery has made more progress in the last hundred years than in all the ages since Hippocrates, the more remote past is full of interest. The dictum of Mr. Henry Ford, of Detroit, proclaims that "all history is bunk", but probably we have more need today than at any period of the world's history to agree with Andrew Lang that "the little present must not be allowed wholly to elbow the great past out of view".

The study of the history of surgery will help surgeons to absorb unconsciously from its proud records a high standard of ethics. It should also imbue us with moderation in esteeming the value of our work and of ourselves.

[1] Read at the Annual Meeting of the Royal Australasian College of Surgeons, Adelaide, March, 1934. The address was illustrated by lantern slides.

It may help our humility to recall, at times, that we have medical records dating back to 2500 B.C.; that asafœtida, henbane, myrrh, and camomile were in use before the Christian era; that the laryngologists then made up their lozenges with liquorice even as they do today; and that the comforting poultice has soothed many a Babylonian whitlow, although we have abandoned the custom of ordering the patient to eat it after application. For at least three thousand years man has unloaded his colon with castor oil and aloes, has been helped to cough up his catarrh with squills, has balanced his acidity with bicarbonate of soda, and has eased his pains with opium. (St. Clair Thomson.)

The fig, the syrup of which is used today to titillate the bowel of the young, was used by the children of Israel as a poultice.

That there is nothing new under the sun explains the eternal applicability of Holy Writ. As a description of a surgeon of the present day cleansed, girt and garbed to perform an operation it would be difficult to improve on the following verse from Leviticus.

He shall put on the holy linen coat and he shall have the linen breeches upon his flesh, and shall be girded with a linen girdle, and with the linen mitre shall he be attired; these *are* holy garments; therefore shall he wash his flesh in water, and *so* put them on.

In the formula which I used in admitting the new Fellows this evening you may perhaps have noticed that I expressed the hope that in addition to practising the art, they would also seek to advance the science of surgery. The equivalent of this modern aspiration occurs, however, as a Brahman precept two hundred years before Christ. It runs:

He who is trained only in theory and is not experienced in practice knows not what he should do when actually dealing with a patient. On the other hand a surgeon who is educated in practice alone and knows nothing of science will not earn the respect of better men.

Coming to the later, but still remote, times of the fifth to the twelfth century, we find that surgery was in the hands of the monastic orders. At the Council of Tours the bull of Pope Alexander finally divorced the church from surgery, proclaiming that it was profane for the educated members of the church to soil their hands with blood. Prior to this the barbers proper had been little more than the servants of the church. Their duties largely lay in shaving the heads of and in bleeding their clerical superiors. What more natural when the rift came between the practice of medicine and the art and craft of surgery, than that the monks should importune the barbers to carry on the discarded work. From being illiterate barbers and shavers, the scope of their practice enlarged and the much despised barber surgeons gradually raised their status from what was in truth a lowly beginning. In London in the early part of the fourteenth century, the surgery was shared by the Company of Barbers and the Guild of Surgeons. Licences to practice were granted by the Lord Mayor on condition that "they should well and truly serve the people in their cures and report to the Lord Mayor and Alderman any surgeon neglecting his patients".

One would have expected the Companies of Barbers and Surgeons to drift apart, but on the contrary in 1540 they amalgamated and formed the powerful United Barber-Surgeon Company. This joint company lasted for two hundred years. Its local habitation in Monkwell Street is still extant and is one of the architectural gems of the City of London. At the Centenary banquet of the Royal College of Surgeons of England in the beautiful hall of the Middle Temple, at which I was privileged to be present in 1900, a silver gracecup was on the table. This choice possession of the Barbers' Company was presented to it by Henry VIII in 1540 in commemoration of the union of the Barbers with the Surgeons. Pepys's Diary contains a reference to this cup in the following words:

Among other observables at Chyrugeons' Hall we drunk the King's health out of a Gilt Cup given by King Henry VIII to the Company, with bells hanging at it, which every man is to ring by shaking after he hath drunk up the whole cup.

The union of the two companies, after an existence of two hundred years, was dissolved in 1745. Those who congregate with barbers are likely to be shorn, and so we find that all the surgeons received as alimony was the endowments of the Arris and Gale Lectures, which are still given at the College of Surgeons. While the union lasted, the Barber Surgeon Company was liable to be called upon to appear in the Lord Mayor's Show. On one occasion it was ordered to provide "twelve of the most grave and comeliest persons apparelled with velvet coats, sleeves of the same and chaynes of gold, to attend the Lord Mayor on horse back". Municipal control of surgeons, in a way, still exists in this city, and I am sure that twelve of Adelaide's gravest and comeliest surgeons would, in their motor cars, willingly attend the Lord Mayor on his assumption of office, in return for the privilege of parking their motor cars when, where, and as long as they liked.

Following the final separation in 1745 a corporation of surgeons was formed. This new society was housed in the Surgeons Hall in Old Bailey, convenient to Newgate Prison, for the supply of anatomical material was provided by the bodies of executed criminals. For reasons which seem trivial the company was dissolved after an existence of years. The surgeons then migrated to Lincoln's Inn Fields and applied to Parliament for a new Bill. This was rejected on the ground that the bodies of criminals could not be conveyed such a long distance as from Newgate to Lincoln's Inn Fields, a distance of about a mile. However, three years later, in 1800, the Royal College of Surgeons of London, now of England, was founded. Several years prior to this, Royal Colleges of Surgeons had been established by charter in Edinburgh and Dublin. The history of their genesis is not unlike that of the English College. I will not weary you with it, but will skip a century and refer to more recent events.

It was no doubt the standing of the Royal Colleges of Surgeons of the United Kingdom which led to emulation by the surgeons of the United States of America, and to the formation of the American College of Surgeons in 1913. In that year Sir Rickman Godlee, Bart., President of the Royal College of Surgeons of England, and nephew of Lord Lister, took part in the proceedings of inauguration. Later, as a further evidence of goodwill, a great mace was presented to the American College by the consulting surgeons of the British Armies in the Great War. This sympathetic regard of British surgeons in general, and of the old College for the

new, in its turn led certain Fellows of the American College to interest themselves in the creation of a college of surgeons in Australasia. To the development and birth of this I am now come.

The formation of an Australasian College of Surgeons was first suggested by Sir Louis Barnett, Professor of Surgery in the University of Otago, New Zealand. In a letter addressed to the Section of Surgery of the Australasian Medical Congress held at Brisbane in 1920, he urged that action should be taken to obtain a higher grade of efficiency in surgical work and to provide for the bestowal of some hall mark on the efficient. His proposal was rejected, it being held that the formation of Sections of Surgery within the Branches of the British Medical Association in the several States would secure the first of his objects—a higher grade of efficiency in surgical work. As a hall mark it was felt that the Masterships of Surgery in the Universities of Australasia were adequate.

Four years later Dr. William Mayo, of the famous Mayo Clinic, and Dr. Franklin Martin, the founder of the American College of Surgeons, at a meeting in Melbourne outlined the ideals, aims and objects of the American College. This stimulus led to the revival in 1926 of the proposal to form an Australasian college.

Forty of the senior surgeons were selected as Founders of the College, and the late Sir George Syme was provisionally appointed President. In 1927, at Dunedin, the Founders elected the first Council of the College, and this body unanimously elected Sir George Syme as President.

His Excellency the Governor-General, Lord Stonehaven, honoured the College by his presence at the first meeting at Canberra in 1928, when the late Sir Neville Howse announced that the Government of the Commonwealth had set apart a site in the capital for a college building. The Royal College of Surgeons of England sent an address of goodwill and expressed the hope "that the foundation of the College may tend to confirm and strengthen the bonds that already unite the medical profession in this country and in the Dominions beyond the seas".

In 1930 His Majesty the King was graciously pleased to approve of the prefix "Royal" for the title of the College, and in the following year the College of Heralds granted armorial bearings, crest, supporters and motto. Two years ago the Council of the Royal College of England most generously presented this College with the very beautiful mace on the table before you, and sent one of its members, Mr. C. H. Fagge, to make the presentation.

I have related to you how the College came into being. Let me now state its chief objects.

The first and most important is to cultivate and maintain the highest principles of surgical practice and ethics. To show just what this entails I cannot do better than quote what Guy de Chauliac, a famous surgeon, wrote centuries ago.

> The conditions necessary for the surgeon are four: First he should be learned; second he should be expert; third he must be ingenious; and fourth he should be able to adapt himself. It is required for the first that the surgeon should know not only the principles of surgery but also those of medicine in theory and practice; for the second, that he should have seen others operate; for the third, that he should be ingenious, of good judgment and memory to recognize conditions; and for the fourth that he should be adaptable and able to accommodate himself to circumstances. Let the surgeon be bold in all sure things, and fearful in dangerous things; let him avoid all faulty treatments and practices. He ought to be gracious to the sick, considerate to his associates, cautious in his prognostigations. Let him be modest, dignified, gentle, pitiful and merciful; not covetous nor an extortionist of money; but rather let his reward be according to his work, to the means of the patient, to the quality of the issue and to his own dignity.

Similarly, the words of William Bulleine, who lived in the sixteenth century, are quaint but true.

> He (the surgeon) must begin first in youth with good learning and exercise in thys noble arte, he also must be cleanly, nimble handed, sharp sighted, pregnant witted, bolde spirited, cleanly apparailed, pitefull harted, but not womenly affeccionated to wepe or trimble, when he seeth broken bones or bloodie wounds, neither must he give place to the crie of his sore Patiente, for soft Chyrurgiains maketh foul sores. Of the other syde, he maie not plaie the partes of a Butcher to cutte, rend or tear the body of mannekynde.

Prior to the discovery of anæsthesia it behoved the surgeon to be as gentle as possible in his manipulations. Nowadays there are surgeons who forget that although the patient under an anæsthetic is oblivious of being handled roughly, his palpitating tissues are sensitive to any rude assault. Lord Moynihan condemns the tearaway surgeon and extols him who deals tenderly with his patient, in the following passage.

> There are surgeons who operate upon the principle of savage attack, and the biting and tearing of tissues are terrible to witness. These are they who operate with one eye on the clock and who judge the beauty of any procedure by the fewness of the minutes it has taken to complete. There are other surgeons who believe in the light hand, who use the utmost gentleness, and who deal lovingly with every tissue they touch. The scalpel is indeed an instrument of most precious use—in some hands a royal sceptre, in others but a rude mattock. The perfect surgeon must have the "heart of a lion and the hand of a lady"; never the claws of a lion and the heart of a sheep.

To those who comprise this audience, and indeed any audience, the second object of the College is as important as the first. It reads:

> To arrange for adequate post-graduate surgical training at Universities and Hospitals and to conduct examinations for admission to Fellowship.

In regard to this let me again quote Lord Moynihan.

> Surgery today is being practised by too many lighthearted and incompetent surgeons who have neither sought in due service to acquire a mastery of their craft, nor have learned from the experience gained by long association in hospital work when an operation should be done, when left undone, how made safe, how made to fall lightly upon a patient, already afflicted, it may be by mental no less than physical distress.

Professor Wood Jones passes similar strictures in his charming book "Unscientific Essays".

> In the days before these gifts (that is asepsis and anæsthesia) were given the master surgeons were the elect. Only the few were fitted for the work, and they were fitted by reason of their practised dexterity, their knowledge, their judgment, their readiness of resource, and by a hundred fine brave manly qualities that have made them great men. But today thanks to the dual blessing of anæsthesia and asepsis, every man determined to be a doctor rather than a linen draper, is able to obtain a patient and may tamper with the innermost secrets of the human body to the end that he may effect a cure. It is made easy for the sons of Æsculapius to take too much upon them.

Lord Moynihan insists that in addition to skilled craftsmanship and the many virtues which are necessary in the surgeon, certain spiritual promptings are called for.

Surgery is not only a craft, though craftsmanship of a high order is essential. It is a profession to which a man should feel not only complete devotion but also a sense of special dedication. Its practice requires the most flawless integrity in thought and act. No one acquainted with the truth can deny that far too many operations nowadays are done by those who having perhaps a measure of success in the trivial cases (none are trivial to a patient) rashly embark upon procedures which they are not fully competent to undertake, and the good repute of surgery is wounded.

It is the object of this College to insure, by the training which it prescribes, that its Fellows to be shall be surgeons, not operators. The distinction is a real one, not understood of the laity. Rutherford Morrison, the great Newcastle surgeon, now living in retirement in the North of England, likens operators to the ordinary golfer. He writes:

Operations are now so safe that there is a general belief that it does not much matter who performs them. Nothing could be further from the truth. Operators are like golfers. Many can play *at* golf after prolonged instruction and practice, but few can ever play golf. Unless the golf is in, it can never come out. There are many operators but few surgeons. In addition to natural gifts a surgeon requires prolonged hospital experience to enable him to get out of any tight corner with the greatest advantage to his patient and to his own satisfaction.

This College has taken steps to assist those who aspire to its Fellowship to obtain that adequate and special training in surgery which is a necessary qualification for it. In view of the fact that it is now necessary for a candidate to possess a senior surgical qualification, the Council has been instrumental in achieving almost complete uniformity in the regulations governing admission to the senior surgical degrees in the Australian Universities. The Council has maintained the closest possible association with the Royal College of Surgeons of England. That College has already held one Primary Fellowship examination in Australia, and at the end of this year will conduct another. The period of training of those who are candidates for the Fellowship of the Australasian College must extend over a minimum period of five years, most of which time must be spent in gaining practical experience in hospital.

The question of operations being performed by the general practitioner demands a word. It is not the policy of this College to object to a general practitioner performing an operation himself, provided he is quite clear as to the possible result. If he is not clear (I quote from and am in entire agreement with Kocher, the famous Swiss surgeon), his conscience ought to lead him to consult an experienced surgeon in order to gain a clear idea of the indications, dangers and technique of the operation. The doctor is most to be commended who establishes a correct diagnosis at once, and regardless of other considerations sends his patients for early operation to the place where there is the best prospect of a permanently successful result. He can then consider that he has been the chief cause of the success, and he is certainly more to be congratulated than the man who, unconscious of his limitations, cannot resist the temptation of trying to prove his ability as an operator under conditions where the requisites for success are unattainable.

Under objects of the College on which I have only time to touch are the promotion of research in surgery. One who held high office in the College, the late Mr. Hamilton Russell, a true disciple of Lord Lister, has set an example for all to follow. The family of the late Sir George Syme have generously endowed a "Syme Surgical Research Scholarship", and the late Dr. Gordon Craig made a bequest of a most munificent nature for the purposes of research.

The College is also keenly anxious to promote the practice of surgery under proper conditions by securing the improvement of hospitals and of hospital methods, and for this purpose has appointed special Hospital Committees. Finally, it must be borne in mind that Australia and New Zealand are jointly and equally concerned in the future of this College. One of the objects for which it exists, therefore, is to bring together periodically the surgeons of Australia and New Zealand for the scientific discussion and practical demonstration of surgical subjects. For such and other purposes it is necessary that the College should have a habitation of its own. Notwithstanding the gift of a site in Canberra, Melbourne offered advantages compelling its selection. On the fine site granted on most generous conditions by the Government of Victoria, a building not unfit to rank with the homes of the Colleges of Surgeons of the world is rising from its foundations. It is hoped that His Royal Highness Prince Henry will lay the foundation stone. It is known that Sir Holburt Waring, the President of the Royal College of Surgeons of England, will open the College next year. In this address I have tried to trace the steps by which the notable and, I am emboldened to say, noble traditions of surgery have been achieved. May we in whose hands they lie in the Dominions beyond the seas prove worthy of our inheritance and add to it truly and well.

THE TREATMENT OF EMERGENCIES IN CARDIAC DISEASE.[1]

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THE subject of this paper is one in which every practitioner is interested, as he may meet one of the conditions discussed at any time. He must make up his mind straight away and act, as there is no time to be lost. If he procrastinates, the patient may die. The emergencies met with in a medical practice are just as urgent and important as those met with in a surgical practice.

As you all have had experience in the majority of these conditions, you will be able to criticize my own ideas of what one should do, and may probably be able to suggest something better. It is with this object that I have written this paper, so that we can find out what is best for our patients.

Acute Congestive Heart Failure.

The first emergency that I will discuss is that of acute congestive heart failure occurring during failing compensation.

[1] Read at a meeting of the Section of Medicine, New South Wales Branch of the British Medical Association, on June 7, 1934.

Here we generally have a patient, middle-aged or elderly, who usually has been ill for some time with dyspnœa on exertion and some œdema, but during the last few days has become much worse. Our patient is sitting propped up in bed, often leaning forward over a pillow, and gasping for breath. The face is cyanosed and the hands are often œdematous. There is generally an oxygen cylinder alongside the patient, and he has a catheter in his nose through which oxygen is passing. These patients generally resent an oxygen tent.

He has been digitalized, alcoholized, morphinized, and oxygenized, with some relief to his feelings, but little to his general condition. On looking carefully at this patient, the veins will be found bulging in his neck, and sometimes in his hands as well, if the œdema is not too great. These veins are asking to be opened. The treatment here is venesection. Take away a pint of blood; it is of no use taking only four or five ounces. Bleeding seems to be a lost art. I have seen numerous medical men trying to bleed a patient by means of passing a needle into a vein and trying to withdraw a large quantity of blood. This way is very often unsuccessful. After a while the blood clots in the needle and it will not run. The proper way to bleed a patient is as follows. If the veins in the arm are markedly distended and clearly visible, a large needle may be used. A small needle is useless, for, if it does not clot up, it will take a long time to draw off a pint of blood, and the patient will become considerably distressed and restless. The better way is to open the median basilic vein and let the blood run into a basin, when a pint can be withdrawn in a few minutes without distress to the patient. Furthermore, the veins in these cases are generally buried in œdema, and it is difficult to get a needle into the vein.

A local anæsthetic is injected over the median basilic vein and an incision is made, and a threaded aneurysm needle is passed under the vein. The needle is now withdrawn, the thread being left behind, and a V-shaped incision is made in the vein with a sharp pair of scissors, and the blood allowed to pour out. When the requisite amount of blood has been withdrawn, a pad and strapping may be placed over the wound, but sometimes it is necessary to suture the skin.

How further can we help our patient? We have relieved the congestion on the right side of the heart and reduced his blood volume, and the heart now wants oxygen and food. Oxygen may now be supplied by inhalation, and food by means of insulin and glucose. For the last two years or so I have been treating my patients suffering from severe heart failure with insulin, five units, and glucose, 10 to 15 grammes, three times a day, in addition to the ordinary treatment, and I feel in my own mind that I have tided a few patients over a critical period with the aid of this method.

Œdema.

You will meet from time to time cases of cardiac œdema which will not respond to ordinary methods, such as *pil. diuretica*, "Diuretin", "Theocin", "Novasurol", with or without ammonium chloride, *et cetera*. The patient's condition is becoming desperate and the only way out of the difficulty seems to be to make incisions in the legs and let him sit up and drain. This is a messy business for the patient, and it will be some time before the wounds heal. In the meantime the dressings round his legs are constantly wet and sodden. These patients are nearly always relieved by ammonium chloride, given before the injections of "Novasurol" or "Salyrgan" in adequate doses. The dose of ammonium chloride recommended by the makers of these drugs is not nearly enough. To get the full effect of "Novasurol" or "Salyrgan" a mild acidosis must be produced, and this is obtained by giving 6·0 to 8·0 grammes (90 to 120 grains) of ammonium chloride in 60 or 90 cubic centimetres (two or three ounces) of water by the mouth twenty to thirty minutes before the injections of the diuretic. If this is done, diuresis, which has not been produced before, will very often become profuse, and I have seen the amount of urine rise to 7·0 litres (230 ounces) in the twenty-four hours, when "Novasurol" given without the large doses of ammonium chloride has produced but little effect.

Acute Pulmonary Œdema.

In acute pulmonary œdema our patient is usually middle-aged or old, of the arteriosclerotic or hypertensive type, yet I have seen the condition occur in a jockey boy of about fifteen years of age. The patient generally has been ill or seedy for a little time before he is attacked by this condition.

Those of you who have seen this condition in its severe form, where the patient is coughing up large quantities of fluid, often frothy and sometimes blood-stained, and is in great danger of being drowned in his own fluids, will realize that few conditions require more urgent treatment. I have seen a patient cough up two or three pints of frothy serum in three or four hours. The treatment that I have found successful is the immediate injection of morphine, 0·015 gramme (one-quarter of a grain), atropine, 0·43 milligramme (one one-hundred-and-fiftieth of a grain), and strychnine, 2·2 milligrammes (one-thirtieth of a grain), combined with bleeding to the extent of one pint, may be given if the veins are engorged. Oxygen is seldom on hand when required; but if it is, an oxygen tube may be held in the mouth of the patient and the oxygen inhaled. Inhalation by any other means seems to be too distressing to the patient. Having relieved that attack of pulmonary œdema, remember that it has a tendency to recur, so be on your guard after about four to five hours. Should it show signs of returning, give a further injection of morphine, 0·01 gramme (one-sixth of a grain) with the atropine and strychnine.

Simple Tachycardia.

Patients often complain that they are awakened at night by attacks of palpitation, which often cause them considerable alarm. This condition is

generally relieved by sipping, not drinking, very hot water. I instruct them to keep a "Thermos" flask of hot water alongside the bed, together with a cup and a teaspoon, and when the attack comes on, to sip this. As you are aware, when swallowing takes place, the vagus is stimulated and so slows the heart.

Paroxysmal Tachycardia.

Paroxysmal tachycardia, you know, comes on like a shot from the blue and stops just as suddenly. Often the patient seems in quite good health, and for apparently no reason at all the attack comes on, and this may last only a few seconds, but sometimes for days.

In mild cases, slow, deep breathing with the arms and head extended often stops the attacks. If this fails, tickling the pharynx to produce vomiting occasionally gives relief. I have never found pressure on the eyeballs do any good. Pressure on the *sinus caroticus*, especially on the right side, often gives relief, and a patient can be taught to do this himself. The pressure should be made below the angle of the jaw, over the carotid artery, for about ten to twenty seconds. According to Lewis, this procedure is without danger. In the more severe attacks, when the patient becomes cyanosed, breathless, and profoundly ill, with symptoms of cardiac failure, which may occur after the attack has lasted several hours or a few days, I have found quinidine given by the mouth, after a few doses, succeed in stopping the attack. Start with 0·3 gramme (five grains) and increase by 0·06 gramme (one grain) every four hours, that is, the second dose should be 0·36 gramme (six grains), the third 0·42 gramme (seven grains), and so on. Stop the quinidine if it produces any symptoms of vomiting, diarrhœa or vertigo, or when the total dosage reaches 1·8 to 2·1 grammes (thirty to thirty-five grains).

It is advisable, if the attack has been relieved, to continue the quinidine in small doses of 0·09 to 0·18 gramme (one and a half to three grains) two or three times daily for some time, providing it does not cause the patient any distress.

In cases of great urgency I have tried quinine, 0·3 gramme (five grains), intravenously without success, when 0·65 milligramme (one one-hundredth of a grain) of strophanthin, on the suggestion of Dr. S. A. Smith, relieved the condition. This was a very unusual case of gumma of the heart. I have never given quinidine intravenously, but, should occasion arise, I would not hesitate to give 0·18 gramme (three grains) by this means.

In place of strophanthin or "Ouabain" we have now at our disposal a new crystalline glucoside made from digitalis, namely, digoxin, which seems to be more reliable and safer than the strophanthins. This may be given intravenously in doses of 0·75 to 1·0 milligramme (one-eightieth to one-sixty-fourth of a grain), dissolved in 8% alcohol in saline solution, 0·4 milligramme of digoxin being equal in cat units to one cubic centimetre of standard tincture of digitalis.

After intravenous injection the fall in ventricular rate begins in five to ten minutes and reaches its full extent in one to two hours. This drug is made by Burroughs, Wellcome and Company. If possible, take an electrocardiograph and find out whether the tachycardia is ventricular or auricular in origin. This procedure will be of great use, for digoxin is indicated in the auricular type, while quinidine is indicated in both types, but is more useful in the ventricular form.

Paroxysmal Fibrillation.

Should fibrillation make its appearance while attending a sick patient, quinidine 0·18 gramme (three grains) may be given three times a day. This drug will often stop the fibrillation, but great care must be taken, if the fibrillation does not stop after a few doses, to see that the ventricular rate does not rise too high. Remember that quinidine slows the auricle but increases the conductivity of the auriculo-ventricular bundle. Now the auricle in fibrillation is showering impulses on to the auriculo-ventricular bundle at the rate of 300 to 400 per minute. If the conductivity of the auriculo-ventricular bundle is increased there is a chance of the ventricle being stimulated too fast; so if the ventricular rate starts to get too rapid, give a big dose of digitalis straight away. If the patient has had no digitalis, give 12·0 to 14·0 cubic centimetres (three to three and a half drachms) in one dose. If there has been previous administration of digitalis, go more carefully and give four cubic centimetres (one drachm) every six hours until the ventricular rate is lowered. It is in cases like this that digoxin is very useful. Here we may give 1·5 milligramme of digoxin by the mouth, when a fall in ventricular rate will begin in one hour and reach its maximum in six to seven hours, or if the rate has risen very fast, 0·75 to 1·0 milligramme of digoxin intravenously is indicated, providing that the patient has had no digitalis previously.

There is a rule to remember when using quinidine: Never give it to a patient suffering from advanced heart disease.

Bradycardia: The Stokes-Adams Syndrome.

In the Stokes-Adams syndrome our patient, who is suffering from a marked degree of heart block with great slowing of the ventricular rate, is seized with attacks of unconsciousness, which may be followed in a few seconds by twitchings and even epileptiform convulsions.

This condition calls for no emergency treatment, except when the attacks recur at frequent intervals. I have seen a patient with as many as three to four attacks an hour. In these cases two drugs may be useful: ephedrine, 0·24 to 0·3 gramme (three-eighths to one-half of a grain), two to three times a day, and atropine in large doses, 2·2 milligrammes (one-thirtieth of a grain) given hypodermically, may increase the number of ventricular beats per minute.

Cardiac Asthma.

The emergency treatment in cardiac asthma, as you all know, consists in giving morphine and oxygen, but care must be taken to diagnose between ordinary bronchial asthma and cardiac asthma. Remember that bronchial asthma is an expiratory dyspnœa, that is, the patient can get his breath quite well, but cannot get rid of it, while cardiac dyspnœa is a general respiratory discomfort.

Angina Pectoris.

There is no need to discuss the emergency treatment of *angina pectoris*, but personally I prefer trinitrin to amyl nitrite, especially if combined with menthol. Such a tablet is made by Burroughs, Wellcome and Company, called "Tabloid Trinitrin Co". It is most important that the tabloids be chewed up and kept in the mouth and not swallowed whole. William Evans and Clifford Hoyle have shown that trinitrin is very easily and quickly absorbed from the mouth, and that its effect is much quicker and greater than when it is swallowed. In the event of there being none of the usual drugs available for the relief of this condition, neat brandy or whisky in 15 cubic centimetre (half ounce) doses is often useful.

Coronary Obstruction.

You have all seen cases of coronary obstruction in which there are collapse, imperceptible or rapid, thready pulse, and an appalling fall of blood pressure, generally accompanied by agonal pain, mostly over the heart, but occasionally in the abdomen.

You will have noticed that there are two types of coronary obstruction, one accompanied by pain and the other without pain.

The treatment in the type accompanied by pain is morphine, and it is no use playing about with small doses. Start with 0·024 gramme (three-eighths of a grain) for small people, and 0·03 gramme (one-half of a grain) for big people, and repeat 0·008 gramme (one-eighth of a grain) every twenty-five to thirty minutes up to 0·06 gramme (one grain). In the meantime it is just as well to anæsthetize the patient slightly with ether. I prefer this to chloroform, as ether has fifty-two times less toxic action on the heart muscle than chloroform; 5% carbon dioxide and oxygen should be administered if there is any respiratory distress or failure, as the big doses of morphine may unduly depress the respiratory centre. In non-painful coronary obstruction morphine is also necessary, but not in such big doses.

Now how else can we help our patient? The coronary circulation consists of three types: (i) the coronary arteries, which anastomose with each other tremendously, and as we get older they anastomose much more; (ii) the Thebesian vessels, where the blood is pumped directly into the heart muscle from the chambers; (iii) the direct opening of the coronary vessels into the heart chambers themselves, where the blood can go directly into the vessels, other than by means of the aorta.

Concerning the Thebesian vessels, we have no knowledge of their innervation, but only of their function. We have definite knowledge that we can increase the coronary circulation by dilating the coronary arteries with the aid of certain drugs. These drugs are caffeine, theobromine, theophyllin and "Lacarnol". The coronary circulation is increased 3% by caffeine, 25% by theobromine, and 45% by theophyllin. As you see, caffeine has but little effect, and I have found that it is almost useless in cardiac affections, but not so with "Diuretin" and "Theocin". "Diuretin", by the way, is a salicylate of theobromine, while "Theocin" is theophyllin sodium acetate.

Here we have drugs that may open up the collateral circulation and so help to nourish the damaged part. We therefore do good by administering either "Diuretin", 0·3 to 0·6 gramme (five to ten grains), or "Theocin", 0·12 to 0·18 gramme (two to three grains), three times a day, provided that the coronary arteries are dilatable, that is, if there is not a very advanced condition of arteriosclerosis. I have never liked to push trinitrin in these cases, as I consider its action too transient and too sudden.

Tapping the Pericardium.

Tapping the pericardium is seldom required, except when there are marked cardiac distress and failure due to the pressure of the fluid in the pericardium. I have often found it very difficult to get into the pericardial sac. I have tried practically all the methods of entering the pericardium through the front of the chest; and I have found them exceedingly distressing to the patient, and more times than not the heart has been penetrated and only blood withdrawn. As you are aware, when there is a great accumulation of fluid in the pericardium there is a dull patch with tubular breathing at the angle of the left scapula. If this area is penetrated, you will find that you first perforate the pleura, then you feel the needle passing through the lung until the end of the needle comes up against an obstruction. Penetrate this and you will be in the pericardial sac, from which the fluid can be withdrawn easily, and the whole procedure causes very little pain or distress, differing markedly in this manner from the anterior route method.

The few patients in whom I have found it necessary to tap the pericardium have all been children or young people.

Conclusion.

The subject matter that I have just been talking about is culled almost entirely from my own experiences in practice. The only drug which I have not had much experience of is digoxin, as this has been a short time only on the market.

I hope that I have not wearied you with such a large accumulation of facts. A large amount of facts is hard to digest, so I have made my paper fairly short in order that you might not get mental indigestion from it.

THE CAUSE OF DEATH: AN ABSTRACT OF 500 CONSECUTIVE AUTOPSIES.[1]

By J. V. DUHIG, M.B. (Sydney),
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AFTER a long experience of *post mortem* work, I have come to the conclusion that clinical medicine must be the most difficult of all the arts. It has been my duty to look inside the clinical material that my colleagues have handled, in now nearly two thousand cases, and when I saw how difficult accurate diagnosis must necessarily be, I very soon resisted all temptation to be scornful of my colleagues' mistakes and failures. Whatever the mistakes or failures, they were at least seldom due to lack of care or reasonable skill and knowledge, but were inherent in the case, as when a cancer of the pancreas first shows itself by a swollen supraclavicular gland. This paper is a review of five hundred consecutive autopsies and an attempt to indicate to the general practitioner what to look for in cases of sudden death or very serious illness, and I want to offer a word on death certification.

The work on which this paper is based was done by me as Government Pathologist for Queensland, and the cases studied are therefore of a special type, about which little was previously known beyond facts within the casual knowledge of the police or obtained by them after a usually rather hurried preliminary inquiry. On occasions, a rapid decision involving the liberty of suspects has had to be made on very slender external evidence, and in a delicate situation of this sort the medical man must be very certain of what he is doing, and not neglect to get all the information by inquiry and reading, very quickly, of course, that will lead to a sound decision. I find it is not much good trusting to memory of what an authority says—it is necessary to consult the book.

The Statute under which I work is entitled "An Act to Consolidate and Amend the Law relating to the Holding of Inquests of Death and Inquests concerning Fires", shortly known as the *Coroner's Act* of 1930. The Act has been slightly amended by regulations made duly by the Minister, and is likely to be drastically amended in the near future, but only in so far as the strictly legal provisions are concerned. The clauses which affect medical men seem to me to be quite adequate. They are:

5. (1) Every Coroner shall have jurisdiction throughout Queensland to inquire into the manner and cause of the death of any person who—

(*a*) Is killed; or
(*b*) Is found drowned; or
(*c*) Dies a sudden death of which the cause is unknown; or
(*d*) Dies under any suspicious or unusual circumstances; or
(*e*) Dies while under an anæsthetic in the course of a medical, surgical or dental operation or operation of a like nature; or
(*f*) Dies but no certificate of a medical practitioner has been given as to the cause of death; or
(*g*) Dies within a year and a day from the date of any accident where the cause of death is directly attributable to such accident; or
(*h*) Dies under such circumstances that, in the opinion of the Minister, the cause of death and the circumstances of such death should be more clearly and definitely ascertained; or
(*i*) Dies in any prison hospital, hospital for the insane, or any institution under such circumstances as to require an inquest in pursuance of this Act or any other Act;
(*j*) Dies, not having been attended by a medical practitioner at any period within three months prior to his death.

5. (3) Subject to this Act a medical practitioner shall not, unless with the consent of the coroner, give a medical certificate as to the cause of death in respect of any death which occurs under the circumstances referred to in paragraph (*a*), (*b*), (*c*), (*e*), or (*j*) of subsection one.

11. The Minister or a coroner may in his discretion order a post-mortem examination of any deceased person for the purpose of an inquest, and the Minister may order the body of any deceased person to be exhumed, and for that purpose the coroner may, with such assistants as he may require, enter and break open any ground, cemetery or place for the purpose aforesaid.

12. Whenever a coroner shall hold an inquest he shall, if there be any reasonable doubt as to the cause of death and it be possible to obtain such testimony, take the testimony of a medical practitioner thereon.

It is quite evident that the coroner may proceed to inquire into a death from violence or in unusual circumstances without first ordering a *post mortem* examination since it is assumed, probably quite rightly, that it is neither the coroner's interest nor his obligation to guarantee the academic accuracy of the Registrar-General's records of deaths. I myself would consider it of the greatest importance that these records be as rigidly accurate as it is possible to make them, but so far the law does not make this care a part of the coroner's duty. Apparently all the law requires is that he shall see that in the circumstances around the death there has been no breach of right or justice. So that, where, in his discretion, the coroner can dispense with a *post mortem* examination which costs the State money, he usually induces some medical man connected with the case to give a certificate of death. From my experience I should say that not more than half the death certificates the Registrar receives would be accurate in every detail as to what is included and what is omitted; so that, though in Queensland undetected crime and serious miscarriage of justice directly resulting from neglect of *post mortem* examination is practically impossible, a good deal of inaccuracy in death certification is inevitable in those cases coming before the coroner without autopsy.

Queensland practice is towards certainty in doubtful cases, but a medical practitioner who is too eager to oblige may mislead the coroner and eventually cause serious embarrassment to litigants. Clinical medicine is such an extremely difficult art, and one is so constantly faced with such extraordinary surprises on opening a body with a seemingly simple straightforward history, that I should counsel my colleagues, should they honestly hesitate to give a certificate to the coroner at his request, to persist in that hesitation until they can either have no doubt as to the very probable cause of death or refuse to certify.

It must be remembered, of course, that a coroner's *post mortem* has for its object to determine the cause of death and to make certain that any breach of the law in the circumstances of the death will be brought to light: it is not meant to afford material for a detailed pathological study.

[1] Read at a meeting of the Queensland Branch of the British Medical Association on May 4, 1934.

Most of the material treated in this paper, however, has afforded me much pathological interest, since, apart from the deaths from suicide and violence, the persons who came to autopsy had been for the most part in reasonably good health and had either never consulted a doctor at all, or not for three months prior to death; they were, in effect, like the man whose condition nobody could diagnose—perfectly healthy at the time of his death. It has been of considerable interest to try to find out why these people died precisely at the time they did. A large number of those examined were old people, and it would be easy to say that senility was the main cause, but senility is, after all, a comparative term, and there does not seem to me to be any special reason inherent in nature why man should terminate his existence between seventy and eighty years of age. I shall return to this question in its proper place later; I merely mention it now to show that there may be still some interest in attempting to decide whether, from the pathological findings, it may be worth while to try to increase still further the average age at death of humans, and whether it is desirable. I shall present the data and indicate what conclusions I think may be drawn from them and leave you to decide whether a longer life than the present average is possible or desirable. I have taken these unusual and, as a rule, tragic deaths simply because they were unusual. The deaths which occur in hospital and in practice can be studied in hospital and registration records, and form the analytical basis of clinical medicine, but little has been said about the special type of case seen on the table of the public morgue.

I have analysed 500 consecutive unselected cases from my records, leaving out of account collections of bones and old skeletons and new-born infants smothered at birth and left in garbage tins and other odd places.

The table shows the type of deaths found in the series and the sex distribution. It will be seen that the classification is of a broad general type so as not to obscure it with too much detail, which will be given in the text under the appropriate rubric.

It will be seen that the males in the series outnumber the females by three to one. This is accounted for by the fact that men commit suicide three times more frequently than women, nearly six times as many men are accidentally killed as women, and three and a half times as many men drop dead or are found dead of heart failure as women. Women, at least in our civilization, do not die sudden and violent deaths to anything like the same extent as men. Why this should be so, I do not precisely know. Perhaps it is that there is a tradition of pride in looking carefully after the health and comfort of women, and when they do die they have had the advantage of having been given the best medical attention available.

Suicide.

In the whole series of 500 unusual deaths there were 106 males and 34 females, 140 in all, or 28%, who committed suicide. This seems to me to be extremely high, though I do not know whether it is abnormal since I have not seen any figures of a comparable kind. My series covers the very worst period in the social history of this community. The incidence of suicide on total deaths arranged according to sex is curiously constant, the male deaths from this cause being 28% of all males examined, and the corresponding female figure being 27%. Whether this correspondence is the result of pure chance, I am not able to say, but I find it of extreme interest as indicating a definite quantitative tendency to suicide throughout the community. From the information of the special kind I am able to gather, I should say that, with some small exceptions to which I shall return in a moment, men are not, as a rule, insane when they commit suicide, women nearly always are. Men commit suicide at all stages of life, in all sorts of ways and for all sorts of reasons, but they do have a reason which seems to me to have been particularly vivid in the minds of the victims. The reason is most often the fear of social disgrace through financial wrongdoing, and not seldom the completely hopeless outlook for the man for whom there is no work and no hope of acquiring the happiness that honestly earned money can buy. Persons who see things in those ways must have vivid imaginations beyond the average, and must be particularly lucid to be able to use their imaginations at all. Of all

Suicide.		Accident.		Cardiac.		Cerebral.		Pulmonary.		Cancer.		Abortion.	Murder.	
Male.	Female.	Male.	Female.	Male.	Female.	Male.	Female.	Male.	Female.	Male.	Female.	Female.	Male.	Female.
106	34	72	13	149	41	24	10	5	8	0	1	1	0	1
140=28%		85=17%		190=38%		34=7%		13=2·6%						

Anæsthesia.		Alcohol.		Renal.		Pernicious Anæmia.		Pancreas.		Birth.	Unclassified.	
Male.	Female.	Male.	Female.	Male.	Female.	Male.	Female.	Male.	Female.	Female.	Male.	Female.
5	4	3	2	2	1	1	0	0	1	1	6	7
9=2%		5=1%		3=0·6%		1		1		1	13=2·6%	

the men who have committed suicide and have been examined *post mortem* by me, only two could be said to be insane in any sense of the word, and both had "got religion" at a revival preacher's meetings. As one of the victims expressed it, he found he was no longer "friends with God". They both acquired a quite unnecessary and probably unreasonable sense of sin, which became such an intolerable burden that it could be discharged in only one way.

Women, on the other hand, tend to commit suicide at the extremes of life, while very young or at the end of middle age. Two very young suicides in my series simply took life too much "*au tragique*", one had gonorrhœa, and the other had had some unfortunate love experience, the true nature of which did not become fully apparent at the inquest. Nearly all the other women suicides had a bad personal or family history and had been under psychiatric treatment at some time and had no special reason for committing suicide.

I think it a terrible thing for a parent to commit suicide, but not such a bad thing for an unmarried person, for the reason that it is good for the race for a person to carry with him a heritable trait which he has not transmitted, and thus save the community the tragic loss of other citizens later. If all persons with a suicidal tendency killed themselves before having offspring, in time the tendency would if not disappear at least be greatly diminished.

The suicide most distinguished for rarity was that of a woman who took cockroach powder containing sodium fluoride, a poison rarely mentioned in medico-legal textbooks. Another curious death was that of a man who stabbed himself in the heart with a boy scout's sheath knife.

In determining the question of whether a death has been due to suicide or homicide, no strict rules can be laid down, each case having to be treated on its merits. In any case, the question is not strictly one for the medical man to decide; that is the duty of the coroner or police, but most often the medical evidence is of paramount importance as a guide to the officers of the Crown. For a time a death, which was the subject of a trial that obtained very wide publicity recently, looked to be much more likely a suicide than a homicide, though the medical data did not allow me to express an opinion either way. I should like to quote a case, however, in which the medical data had to be used to solve the question, and solve it quickly, since the liberty of a subject was at stake.

Suddenly, one peaceful Sunday afternoon, a man rushed to a police station and laid an information that a woman had committed suicide by cutting her throat with a razor in a secluded rural spot, where the informant and the deceased lady had been having a quiet picnic. On seeing the gash in the victim's throat, I surmised that the wound must have been self-inflicted by a left-handed person or homicidally inflicted by a right-handed person. The man was naturally detained in custody, at least until after the *post mortem* examination. His story was consistent and obviously candid. And from the distribution of the blood on the woman's right hand and clothing, I could not possibly come to any other conclusion than that the woman had cut her own throat. With the natural perversity of a woman she had used the razor with her right hand in just the opposite way to that in which a man would use it. The man was quickly liberated and no more was said of the matter.

Accident.

Eighty-five, or 17%, of the deaths were from accident which, when it was not drowning, was due to the action of a high speed vehicle. Of this kind of lethal agent far the most pernicious is the motor-cycle. Nearly six times as many men as women succumb to accident, the reason for this being the obviously much greater exposure to risk.

Apart from these violent forms of death, I have grouped all the others into broad general categories under the rubric of the system of the body involved. by far the most important is the circulatory, there being 190 deaths, or 38%, in the group of diseases of the heart and vessels, by far the largest of all the groups, and this with the suicide and accident groups accounting for 83% of all deaths of which the coroner must take cognizance.

The commonest cause of deaths of all kinds, whether natural or unusual, sudden or long foreseen, is disease of the heart, and this system is by far the commonest involved in sudden death. Strictly speaking the disease which actually kills is disease of the vessels. Very accurate records, even at this late date, are not available for the estimation of the frequency of death from vascular disease of all types, that is, venous thrombosis in all its manifestation as well as thrombosis of the coronary or cerebral arteries. It will be apparent from what I say in a moment that the old dictum "that a man is as old as his arteries" is profoundly true, truer I should surmise than the coiner of the phrase was aware of. From what I have seen of the human body after it has taken all the shocks and encounters of long hard lives, I should say that there is no reason inherent in the structure of the various organs, in their parenchyma or supporting structures, why men should not live to the span of one hundred years, that is, if we believe that life is a boon beyond, say, seventy-five. All that goes wrong in the large majority of the human material I have studied is the vascular system.

Disease of the Heart and Arteries.

Under the heading of disease of the heart and arteries, in which I have also included syphilitic disease, there were 190 deaths, or 38% of all the deaths. Of these 190, 149 were men and 41 women. This unilateral sex incidence is striking. In taking out the records I listed cerebral hæmorrhage under disease of the brain, though strictly it should be included here, but as there are some special problems associated with cerebral hæmorrhage I leave the small group of brain deaths aside for special consideration. The 149 males formed 39% of all the men examined, and the 41 women 32% in their group, so that as in suicide, we may expect a constant proportion in each sex, forming in turn a constant proportion of all sudden deaths to be of this type. The average age at death of the males in this class is 61, and of the females 63·7, a slight, though possibly significant, difference. These averages are high in comparison with the usual expectation of life, but low in comparison with a large number of ages in the whole group, and it is of particular interest to inquire why some people, otherwise normal and sound, should be overcome by vascular changes about, say, fifty years

of age, while in another precisely identical changes do not occur before, say, eighty. It may be a matter of diet or of intercurrent disease which has left no other trace after it has occurred and which cleared in early life.

By far the commonest cause of sudden death is disease of the coronary arteries, and on this subject I should like to offer some general remarks. I have had the opportunity of seeing well over two hundred hearts from subjects who have died of coronary disease, and have intensively studied the pathological anatomy in a few, and I exhibit some stereoscopic radiographs of coronary systems injected with an opaque injection mass. (See special plate.) These I prepared to try to elucidate those very puzzling cases which I shall describe later in which no actual thrombus could be found, but in which the coronary system was very obviously diseased and the history was typical of the sudden anginal catastrophic kind. The pictures show the anatomy of the system very clearly and the grades of change from a healthy patent vessel to complete block high up in the system. The site of the block in a coronary vessel has a profound effect on the subject's future, and I shall try to indicate what conclusions I have been able to draw from the necessarily fragmentary and in detail fairly inaccurate clinical information I have been able to get from the police.

Before going on, however, to a discussion of this aspect of the cause of death, I should like to refer to two points in autopsy work which I have found to be not only of value but which seem to me to be of paramount importance, namely, the degree of calcification or ossification (which amounts to the same thing) of the costal cartilages and the state of the pulmonary artery and (of rather secondary importance) of the pulmonary veins. Before raising the costernal flap to expose the thoracic viscera, I always try the consistence or resistance of the costal cartilages to a sharp fairly heavy knife. After a short experience the amount of calcification of the cartilage can be estimated. The importance of the examination of the pulmonary artery I shall discuss later when I come to speak of the part played by the lungs in cases of sudden death.

I have found that in general the degree of atheroma and of calcification of the arteries runs exactly parallel with the degree of calcification of the costal cartilages. The degree of senility, using that term in a physiological rather than in a purely temporal sense, can be accurately determined by this degree of calcification of the rib cartilages. If a man of forty has rib cartilages more advanced in calcification than those of a man of seventy, then he is the more senile man: he is closer to death, which after all is the test of senility.

In an attempt to make use of this observation clinically, I obtained the kind cooperation of Dr. Markwell in having radiographs made of the rib cartilages of definite heart subjects. The results, for reasons associated with technical limitations, were far too indefinite for any conclusions to be safely drawn. I am sure, however, that refinement in radiological technique will bring the matter into the domain of practical clinical value. I have made sections of rib cartilages to decide exactly why they did calcify in precisely the way they did, which is completely capricious, so far as I can see. If you take an X ray picture of a partially ossified rib cartilage you will find it mottled in a purely haphazard fashion; I mean there is no uniformity as in the ossification of bone in young growing subjects. On examining these foci of calcification, they are found not to be septic emboli, but probably thrombi in occluded vessels. Now the change is not a strictly necessary accompaniment of old age, since the pathologist not seldom finds in very old men, and especially women who die of cancer or some disease not associated with the vascular system, that the rib cartilages cut with the same ease as those of a baby. I find then that the degree of calcification of the rib cartilages is a good guide to the amount of atheroma and calcification one is going to find in the coronary system.

Sudden death or death occurring in a catastrophic way minutes, hours or days after the onset of severe pain and cardiac shock and collapse are due in the large majority of cases to blockage of the coronary arteries, in the large majority of cases blockage of the left anterior branch of the left coronary artery. The onset of the trouble is not always, indeed seldom, directly associated with severe exertion. Often there is a history of exertion some hours before, but in my series it is extremely rare to find that the subject has dropped dead while undergoing severe exertion. About 70% die while asleep or at rest or moving about the house in a quiet way: the frequency of death in the early morning when the subject has just got up or just after breakfast is very striking. This lack of connexion with exertion and work is a fertile source of litigation in the industrial world. It seems to be a tradition (sound or not, I do not know) to expect to associate heart failure with exertion. There seems to be a fairly sound physiological basis for a theoretical opinion that that should be so, but in practice much stretching is necessary to connect the two; and it seems as if physiology might have to be altered in the direction of testing the hypothesis that, so far as exertion is concerned, the heart will not let us do much beyond its capacity to supply energy; that is to say, that, as in the healthy subject whose skeletal muscles tire long before the myocardium, something analogous is to be found in the case of the diseased heart. By all this I mean that I find it hard to understand why exertion should specially tend to produce thrombosis in a coronary vessel, since in the majority of cases of which I am aware, thrombosis occurs quite independently of exertion, while, in fact, the subject is asleep, that is, when the skeletal muscles are at their lowest plane of energy output.

Coronary block, as I have seen it, is roughly of two types, complete and high and resulting in sudden or rapid death, or incomplete and low and resulting in cardiac infarction and subsequent recovery or in cardiac rupture and death. There is a third type, not usually described, which I shall later discuss. The first type is usually a thrombosis high up in the coronary vessel involved, usually the anterior descending, supplying the left ventricle and, be it

noted, both branches of the bundle of His, the right as well as the left, a fact to which I shall again refer. The thrombus is usually just below the origin of the vessel, in which case the circumflex and marginal branches are also involved or, more often, the thrombus is below the origin of the marginal branch of the vessel. The thrombus is invariably formed on an atheromatous plaque, and may take some time to form, varying from a bright red fresh clot to an old hard dry firm organizing thrombus. I have not been able to get a satisfactory history of the symptoms in this latter kind, which is not common, but I should think it would produce increasingly severe dyspnœa, slight at first, but rapidly fatal at the end. The whole outcome depends on the establishment of a collateral circulation, an almost hopeless affair when we consider the anatomy of the part. The case is different when the block occurs low down, again most often in the lower part of the anterior descending branch, usually at the junction of the lower and middle thirds of the course of the vessel, or perhaps a little lower. Recovery from this type is not at all uncommon, as I frequently come across old grey infarcts in the left ventricle and papillary muscles and quite large aneurysms about the apex of the heart. When the block occurs by thrombosis, an infarction takes place before the collateral circulation from the right side and from the marginal artery through its septal branches can come into play. Sometimes the infarcted area stands the strain of the ventricular stroke and, to judge by the number of old infarcts that I find, this is not an uncommon occurrence, or the infarcted area gives way and rupture of the heart occurs. In the present series there were ten cases of rupture of the heart following on infarction and seventeen cases of infarction without rupture. This possibly indicates how often recovery from small infarcts must occur during life.

A third type of sudden cardiac failure is that associated with obvious coronary block and grossly atheromatous vessels, but in which no thrombus can be found macroscopically and in which occlusion of the vessel or vessels by atheroma is not complete, some circulation through them being possible. Histological sections of the terminal branches of the vessels showed no large emboli, and I have come to the tentative conclusion that the sudden and complete cessation of life must be connected in some way with the conduction system of the heart. It must be remembered that the bundle of His is supplied on both its branches by the same artery, the anterior descending coronary, and that occlusion of the vessel by spasm might result in complete and acute bundle block in both the right and left branches; usually, of course, right branch bundle block is only partial. I have not been able to devise a way of testing this hypothesis experimentally, but it is the only rational solution I can think of to explain these curious and inexplicable deaths.

The onset of cardiac failure of this kind is of clinical interest. Almost invariably I get the story that the deceased person complained for a varying time, usually short, though sometimes for days and even for months and, rarely, for years, of pain in the precordial region. That is quite a straightforward history such as one would expect. But not a few also complain of epigastric pain and even of pain in the right hypochondrium suggesting cholecystitis and prompting operation. These people also almost invariably think the trouble is indigestion and take a dose of soda. How they acquired this tendency is a problem I have never been able to solve, except on the supposition that the public learn their physiology from the patent medicine advertisements. Not seldom, too, patients feel an urgent call to stool, and many of those who die suddenly are found on the floor of lavatories.

Syphilitic Disease of the Arteries.

Syphilitic disease of the arteries is usually an aneurysm of the aorta, and that a saccular aneurysm of the ascending aorta. I have seen two dissecting aneurysms, one eventually finding its outlet under the epicardium of the right ventricle. There were 18 cases of ruptured aneurysm, or 3·6%, 14 being in men and four in women. One aneurysm deserves description.

It occurred in a Chinese market-garden laborer, who worked all one hot morning in the garden; he lay down at midday for a rest and died in his sleep. He had just returned from a trip to China where he had gone to consult a fellow countryman believed to be skilled in the cure of disease, but quite unqualified and merely making an easy living out of the credulity of the faithful. My subject had returned quite "cured". The aneurysm had ulcerated through the chest wall and ribs, and was presenting under the skin of the right side of the chest as a swelling eight centimetres in diameter. Under this was the aneurysm which had also ulcerated into the sternum for a depth of two millimetres. The aneurysm, pericardium and left lung were matted into a dense mass difficult to disentangle. The sac had ruptured into the left pleura. On critical examination, the aneurysm was found to be eleven centimetres from above down, eight centimetres from side to side and twelve centimetres from before backwards. The sac was pendulous and the lower part overlapped the right ventricle. Rupture occurred on the left wall opposite the root of the pulmonary artery. The superior *vena cava* was compressed under the posterior wall of the sac to the calibre of about the femoral vein. The pendulous part of the sac contained an enormous laminated thrombus.

This was an heroic aneurysm.

Brain Conditions.

With the exception of one case of polioencephalitis and one of brain tumour, the 34 deaths associated with cessation of brain function were those of cerebral hæmorrhage. These make up about 7% of the total, a surprisingly small number. I do not forget that many attacks of cerebral hæmorrhage are not immediately fatal, and that those likely to succumb to this catastrophe eventually are usually under observation at the time of death, and never come to autopsy. I have, however, in my collection the brain of a man who died of cerebral hæmorrhage which showed the unmistakable large scar of an old hæmorrhage. I have seen but this one case in something near 2,000 autopsies. Of the 34 persons who died in this way 10 were women, nearly half the number of men. This is a much higher proportion of females than in the other groups we have noticed so far. I am in the habit of saying that women tend to die of cerebral hæmorrhage, and men of coronary block.

I have separated vascular disease of the brain from that elsewhere for a special reason. First, congenital defect of the vessels of the brain is not uncommon, and hæmorrhage of the brain or, more

strictly, from the intracranial arteries, is not infrequently due to rupture of a small aneurysm. It has been shown that the vessels in the skull are specially prone during embryological life to defective development, and there is some ground for supposing that these defects may be hereditary. Again, it has been shown that the cerebral arteries, especially those in the brain substance, for example, such as the notorious lenticulo-striate, are very badly supported by adventitia just past the point where a branch leaves a main trunk. These obscure problems of anatomy make the pathology of cerebral hæmorrhage a special study.

It is now the time for me to say that in the two groups of causes of death outside suicide and accident responsible for 45% of all deaths of a sudden or unusual kind, disease of the arteries is fundamentally the fatal lesion, and that disease is atheroma. Arteriosclerosis, I find, comparatively is extremely rare. Atheroma is extremely capricious in its distribution. It may be marked in the ascending aorta or it may be slight, but after forty it is very common in the coronary arteries, especially the anterior descending branch of the left coronary; and the best place to observe it in the whole of the body is the aorta at its bifurcation and the common iliacs. But—and this is a most surprising thing—atheroma in these situations is very rare in syphilitics. In my experience syphilis is extremely rare as the cause of coronary disease, and I am fond of demonstrating the smooth intima of the abdominal aorta at its bifurcation in cases of ruptured aneurysm. A fairly safe rule, apart from coronary disease, is syphilis above the diaphragm, atheroma below it. This is not the place to discuss atheroma in detail, but if we knew its pathology and could prevent it, we would have in our hands the power to prolong life indefinitely.

Lung Conditions.

Only thirteen deaths are recorded under the heading of lungs—five in males and eight in females. I have found fatal tuberculosis only twice, but the number of bodies which do not show signs of old tuberculosis are rare. These signs are principally subapical scarring, more often right than left sided, and of secondary importance is pleural adhesion. I have not kept any special record of these signs, as it was quite apparent at the very beginning that tuberculous infection at some time of life was almost universal. No doubt I have seen many very beautiful, completely unscarred, healthy lungs, but they are a small percentage of the whole.

In hospital practice I find pulmonary thrombosis and embolism so frequent (Belt, of Toronto, reports this complication as being 10% of all hospital autopsies) that I always very carefully open the pulmonary artery and feel up into the two main branches for thrombi. This I have found only twice in the cases of the kind here discussed. This is only to be expected, as pulmonary embolism and thrombosis occurs principally in persons with faliing or sluggish circulation in a state of chronic or temporary invalidism, and this is precisely the type of person whose death does not concern the coroner.

I have seen, in this series, only one case of fatal pulmonary œdema, and I mention it here to show how extraordinarily rapid this complication may be in its onset.

The subject was a young girl of seventeen who was apparently well at her bedtime, 7.30. About 9 p.m. she complained of feeling sick and the ambulance being called, she was taken to the Brisbane Hospital from her home, a distance of about four miles. When the ambulance arrived at the hospital before 9.30 the child was dead and was not taken into hospital. I found her lungs waterlogged, and this with some cerebral œdema was held to be the cause of her death. The trouble was traced to an arteriosclerotic nephritis.

Pulmonary œdema can be very sudden, and not seldom I have found froth about the mouth of people who have died of congestive heart failure following coronary block after a very short illness to be measured in minutes. I should think this a complication to be greatly feared in practice.

Of the other groups shown in the table I have time to discuss only a few.

Abortion.

Most women who die after abortion die in hospital. The only case of this kind coming within this series of sudden death was that of a woman who was aborted one morning and died quite suddenly the following day. The abortionist left no trace of the operation, excepting an empty implantation site, and death was due to pulmonary embolism. At this point I should like to say that I have never had, in all my experience of *post mortem* work, to fall back on a diagnosis of air embolus. It is said to occur, but I have never seen it, and have never felt the need of wanting to look for or find such a thing. I think that if one is careful enough, one will find the pulmonary or cerebral embolus responsible for deaths of this type.

Anæsthetic and Post-Operative Deaths.

I do not know why the coroner is required to take cognizance of anæsthetic deaths. I suppose it is because the law and public opinion are the usual century behind enlightened opinion and have not caught up with anæsthetics. I cannot see how a death from an anæsthetic is different from a death from pneumonia or typhoid fever. But, of course, in a community in which fumigation is not only tolerated but actually encouraged, demanded and enforced, anything comic is to be expected. I should think that an anæsthetist is required to have a considerable degree of skill before he is asked or allowed to give an anæsthetic, and he would rather do anything than kill his patient; the law seems to indicate an attitude quite the opposite on both these points.

Alcoholism.

All I want to say on this point is that alcoholism does not produce cirrhosis of the liver. The best example of cirrhosis I have seen was in the liver of a teetotal old lady. The signs to be looked for in the alcoholic are principally the large greasy liver, rather pale tan in colour and smelling like a tannery (this is unmistakable), the opaque very milky pia-arachnoid and possibly a fatty, flabby heart. The principal poison of this type used in this series is methylated

spirit, sweetened, flavoured and coloured in many cases with aniseed-peppermint lollies. This disguises the flavour of the pyridine with which the spirit is denatured, and makes a palatable drink for those who like this kind of thing. The use of methylated spirit is due to the high price of good potable spirit, most of the victims not being satisfied with beer, and to the accessibility of the spirit in the very numerous and handy grocery shops.

Kidney Disease.

As most of the sufferers from renal disease die in hospital or after being attended by a medical man, nephritis is rarely found in coroners' autopsies. The only cases found in this series were the typical arteriosclerotic kidneys so common at all ages in Queensland. The other kidney case was one of bilateral pyo-nephrolithiasis, quite unsuspected during the life of the subject.

Other Deaths.

The other groups in the table are murder, death being by a gunshot wound through the heart; one case of pernicious anæmia which had been treated unsuccessfully by a herbalist, after a medical practitioner had missed the diagnosis and thus settled any chance we had of catching the quack; one case of cancer of the pancreas, a disease which I have shown elsewhere to be a very difficult one to diagnose; and one case of ruptured *tentorium cerebelli* in a new-born baby.

Conclusion.

The large majority of sudden deaths, then, are due to diseased arteries, the disease being in the large majority atheroma, so that our destiny is balanced on a few droplets of fat.

Acknowledgements.

I wish to thank my assistant, Miss Gwen Jones, for her help in making the injections of the hearts, Dr. Clive Uhr, radiologist, for making the radiographs, and the staff of Kodak Limited, Brisbane, for their technical advice in reducing and mounting the prints.

Reports of Cases.

THE TREATMENT OF POLIOMYELITIS BY DIATHERMY.[1]

By W. KENT HUGHES,
Melbourne.

THOUGH the series of six cases herein described is so small that it is not wise to draw any definite conclusions, yet the results are so encouraging that I bring them forward in the hope that more extensive trials may be made. Perhaps I have been fortunate in my cases in that any extensive sclerosing has been absent, but they are not in any sense picked out for the sake of booming a method which is new to most people, but which has been used and written about by Bordier, of Lyon, for very many years.

Case I.

The first patient was a young woman of twenty who had been a patient of mine at the Children's Hospital fifteen years previously. As the records have been destroyed, no data are available of her condition. She evidently had a severe form of *talipes equino-cavus* (left), for which I operated. When I saw her two and a half years ago she had a very early *talipes equino-cavus* on the right side, with excellent condition of the right muscles. On the left she had slight wasting of the left buttock, 2·5 centimetres (one inch) of wasting of the left thigh 20 centimetres (eight inches) from the anterior superior iliac spine, and 2·5 centimetres (one inch) of wasting of the left calf. Her foot was in good position, except that there was a right-angled contraction of the *tendo Achillis*. By vigorous exercises the right foot was corrected, but no impression could be made on the left *tendo Achillis*, so I lengthened it. After six months, as her condition was not satisfactory and she had to walk a long distance to her school, I obtained her transfer to Melbourne and instituted diathermy treatment to her spine. At the end of six months her improvement was marked. The left buttock was equal to the right, her thigh measurements were equal, and her left calf improved by 9·0 millimetres (three-eighths of an inch). She could walk long distances without tiring, and could play basket-ball with her pupils. This case is well known to the school medical inspectors of Victoria.

Case II.

The second case is that of a boy of twelve years who was sent to me by Dr. Southby, the organizer for the Anterior Poliomyelitis Committee of Victoria. He had an attack of anterior poliomyelitis at ten months. His condition presented the following features: Shortening of the left leg from the knee, 1·25 centimetres (half an inch), right-angled contraction of the foot, complete absence of power in both peronei, the dorsal flexors and the two tibials.

After the application of diathermy to his lumbar enlargement, the peronei both gave evidence of activity after a fortnight's treatment; then there was a flicker of activity in the dorsal flexors. After three weeks the anterior tibial could be felt acting. After five months the peronei are as strong as those of the other foot; the dorsal flexor of the fifth digit is acting strongly. The dorsal flexor of the hallux and the anterior tibial are acting better, but there is no ascertainable response from the posterior tibial. The everted foot can be brought to the mid-line, but not inverted. After eight months there was a flicker of activity of the posterior tibial, and his foot can be inverted just beyond the mid-line. His left foot has increased in length and breadth, and he can now wear in comfort the same sized boot as on the right. The left leg is still 1·25 centimetres (half an inch) shorter, but it is gratifying that increase of shortening has stopped over the last ten months. His *tibialis anticus* has become stronger, but dorsal flexion is poor. I showed this boy last year and you will no doubt note the improvement.[1]

Case III.

The third case is that of a well made boy of three years with a history of slight indefinite illness towards the end of his first year. He had a lot of wasting of the left leg and foot muscles, severe *talipes calcaneus* and flail foot. The long axis of the calcaneus was almost in the same plane as the tibia. There was no apparent activity in any of the muscles of the foot, and the *tendo Achillis* could not be felt. The limb was put up in plaster in plantar flexion with strong traction on the posterior end of the *os calcis*, and diathermy was applied to the lumbar enlargement. The plaster was renewed every ten days or a fortnight, according to its condition. At the end of three weeks the *tendo Achillis* was acting definitely and with slight digital pressure would resist dorsal flexion beyond a right angle. It has always been my custom to treat *talipes calcaneus* by the above plaster method, and I used to be delighted if I could get some return of activity in the *tendo Achillis* in six months. Diathermy

[1] The first six patients described herein were shown at a meeting of the Melbourne Pædiatric Society on April 18, 1934.

[1] Since April 18 growth has taken place in the patient's tibia and fibula (left); there is only 3·0 millimetres (one-eighth of an inch) difference between the two sides.

must be given credit for the quick and satisfactory return of power. At the end of three months there was a definite return of activity of the dorsal and plantar flexors. At the end of six months all muscles show improvement, and he has been walking without plaster or support for a month.

Case IV.

The fourth case is that of a lad, aged twenty years, who had anterior poliomyelitis at five years of age. He was treated at the Children's Hospital for seven years with a deltoid splint *et cetera*. Since twelve he has had no treatment and was advised to have the shoulder joint fixed. His forearm was well developed; the *pectoralis major* was acting feebly and also the biceps; there was hardly any evidence of activity in any other muscles in the vicinity of the shoulder joint. The deltoid seemed to be completely absent, and the head of the humerus wobbled all over the place. Today, after three months, he has shown satisfactory improvement; the pectoral muscles are acting strongly, the *serratus magnus* fairly well, and there is much improvement in the triceps and some in the biceps. The teres muscles have improved very greatly. The deltoid can be definitely felt to be acting. His shoulder joint is less wobbly, but the question of fixation is not yet to be dismissed. His scapula is much wasted; there has been improvement in the rhomboids, but none in the trapezius.

Case V.

The fifth case is that of a girl of seventeen with a history of anterior poliomyelitis at one year. She had a severe *talipes equino-varus* and *cavus* on the left side, and a right-angled contraction on the right, with complete absence of power of the tibials and toe muscles. In spite of elaborate irons, the right medial malleolus almost touched the ground as she walked, or rather wobbled.

There was much wasting of the left buttock and thigh muscles. The right buttock was fairly developed; the rest of the limb was much wasted. The abdominal muscles were very weak, and the spinal muscles deficient. When lying prone or supine, she could not raise her shoulders without using her arms. The left foot was operated upon, and later the right *tendo Achillis*; and diathermy treatment was instituted. Both buttocks are now well formed, the left being almost equal to the right; the thigh muscles have improved very much. The leg muscles on both sides still lag behind, but there is returning power in the foot muscles. Much difficulty was experienced in teaching her to walk properly.

Her abdominal and spinal muscles rapidly improved, and I think they are now stronger than the average, owing to her efforts to strengthen them. In her case, the only one of the six, heliotherapy has been effectively carried out.

Her left foot may want more surgical treatment, and her right foot presents a difficult problem from the weakness of the tibials and the medial ligament of the malleolus.

Her right thigh, measured 15·0 centimetres (six inches) above the patella, was 25·0 centimetres (ten inches); one month later it was 27·5 centimetres (eleven inches); now it is 43·75 centimetres (seventeen and a half inches) in circumference. The left measured 26·25 centimetres (ten and a half inches), one month later 27·5 centimetres (eleven inches), now 35·0 centimetres (fourteen inches). Her calves have increased 9·0 millimetres (three-eighths of an inch) and her *vastus internus* (right) is well developed (see Figure I).

Case VI.

The sixth case is that of a girl of twenty who had anterior poliomyelitis at four and a half years of age. She had severe *talipes equino-varus* with *cavus* on the left side. Her ilio-psoas was the only muscle in good form. Her left buttock and thigh were much wasted, and she could not move any of her toes. The adductors were weak, but acting slightly; abduction was nil. There was no evidence of activity of the quadriceps or hamstrings. She has been under treatment for six weeks. Her left buttock has much improved; her thigh has increased by 6·25 centimetres (two and a half inches); her dorsal and plantar flexors are acting; action of the hallux is just evident. Her trunk muscles were very weak; like the other patient, she could not raise her body without using her hands. She can now raise her body without using her hands.

Figure I, showing patient in Case V.

The *cavus* offered some difficulty—division of the capsules of the joints on the medial surfaces had to be extended well on to the plantar surface. She began walking on crutches four days after operation, with one crutch in three weeks, and in a month with a stick. There is some return of power in all muscles of the thigh and, except evertors and invertors of the foot, some slight return in leg muscles. The hamstrings are lagging behind the other thigh muscles.[1]

Comment.

After forty-four years' experience of anterior poliomyelitis, I have no hesitation in ascribing the satisfactory results obtained to diathermy. The two outstanding features are: (i) the quick return to activity of some of the muscles, which seemed to be merely asleep, and (ii) the progressive increase of activity over many months. I have purposely avoided splints and so miscalled muscle reeducation.

The treatment may be empirical; it is certainly satisfactory. In the case of the child, it surely cannot be psychical; besides, the increase of activity spread over many months completely discounts that theory. Bordier's oldest recorded case was eighteen months after onset. The only new point I have brought forward is in instituting treatment so many years after the attack of anterior poliomyelitis.

[1] On April 18 she could pull 12 pounds with her left leg; today she can pull 28 pounds.

Table showing the pull in pounds of leg muscles on a spring balance.

Side.	May 7, 1934.	May 8, 1934.	May 9, 1934.	May 11, 1934.	May 16, 1934.	May 21, 1934.	May 24, 1934.	May 28, 1934.
Right	10·2	13·0	14·0	16·75	19·00	19·5	20·5	24·4
Left	11·7	13·9	16·5	17·50	19·75	20·0	22·2	23·6

Case VII.

M.T., aged twelve years, who had a three weeks' history, was a patient of Dr. Southby. She was seen on May 5, 1934. She had hyperæsthesia of the thigh muscles on both sides, with some hyperæsthesia of the right calf muscles. There was weakness of the muscles of the lower limbs, especially of the quadriceps. Hyperæsthesia of the spinal muscles with marked lordosis was present. Diathermy was applied at the level of the lumbar enlargement.

On May 6, 1934, the lordosis was almost gone; the legs felt easier. On May 8, 1934, the lordosis was completely gone. No painful sensation was noted anywhere. The patient could not raise her shoulders off the bed.

The pull of the leg muscles on a spring balance is set out in the accompanying table.

An average of three pulls was taken by Dr. Southby.

The spinal muscles recovered more quickly than the abdominal, and today are about normal. The abdominal muscles are improving and the patient can raise herself from pillows; they showed no improvement for eleven days. She began walking on the fifth day after treatment, and by the eighth day could walk quite well.

Comment.

The interesting points about this case are:

1. The rapid recovery of the leg muscles.
2. The almost instantaneous disappearance of hyperæsthesia.
3. The slow recovery of abdominal muscles.
4. That early use of lower limbs in no way retarded recovery.
5. No splinting or massage was used.

TWO CASES OF MENINGITIS ASSOCIATED WITH ACUTE MASTOIDITIS.

By ASHLEIGH O. DAVY, F.R.A.C.S.,

Honorary Assistant Ear, Nose and and Throat Surgeon, Royal Prince Alfred Hospital and Royal Alexandra Hospital for Children, Sydney.

Case I.

K.C., AGED thirteen years, was admitted to the Royal Prince Alfred Hospital on October 11, 1933, with a history of a dizzy feeling in the head with vomiting and slight cough three weeks previously. This was followed five days later by right earache, and then four days later by purulent right aural discharge.

This persisted in an otherwise symptomless period until three days prior to admission, when he developed right frontal headache. During twenty-four hours prior to admission he suffered from vomiting, nausea, generalized headache and giddiness.

On examination his temperature was 37·8° C. (100° F.), his pulse rate 124, and his respiratory rate 30 in the minute. He lay curled up on his right side and looked very ill. Redness, tenderness and slight swelling were present over the right mastoid. Thick purulent discharge came from the right ear. The nose and throat were clear. Slight spontaneous nystagmus to the right was present. Kernig's sign was present and neck rigidity was pronounced. Other systems were clear.

Lumbar puncture yielded two cubic centimetres of thick turbid cerebro-spinal fluid under very low pressure (the fluid was too thick to run fast through the needle). A direct smear revealed Gram-positive diplococci which proved on culture to be hæmolytic streptococci.

Right radical mastoid operation revealed thick pus throughout the mastoid air cells. The *dura mater* of the lateral sinus and of the middle fossa was exposed, the latter widely, and no abnormality was visible to the naked eye. The *dura mater* was not incised. The wound was packed with eusol gauze.

A smear from pus in the mastoid revealed streptococci which proved on culture to be hæmolytic (pure culture).

At the termination of the operation an intramuscular injection of 60 cubic centimetres and an intravenous injection of 30 cubic centimetres of polyvalent antistreptococcal serum (antibacterial) were given.

Twelve hours later the temperature was 39·2° C. (102·6° F.). Lumbar puncture allowed the escape of 15 cubic centimetres of turbid fluid under markedly increased pressure. The patient was drowsy, thirsty, restless, and mentally hazy.

On October 12, 1933, the patient was restless and mentally hazy, but slightly improved. On lumbar puncture 20 cubic centimetres of slightly less turbid fluid, under still greater pressure, were removed. An intramuscular injection of 60 cubic centimetres and an intravenous injection of 30 cubic centimetres of antistreptococcal serum were given.

On October 13, 1933, restlessness was decreasing. In the forenoon lumbar puncture yielded 25 cubic centimetres of cerebro-spinal fluid that was slightly turbid. Ten cubic centimetres of a 10% solution of urotropin and ten cubic centimetres of air were injected intrathecally.

In the afternoon the patient's temperature was 37·8° C. (100° F.). Fifteen cubic centimetres of turbid cerebro-spinal fluid were removed; the fluid was slightly more turbid than that removed in the forenoon. Conversational voice was heard in the right ear at a distance of one foot. The cerebro-spinal fluid contained 5,400 polymorphonuclear cells per cubic millimetre. In a smear polymorphonuclear cells were profuse. No organisms were found. Culture was sterile.

On October 14, 1933, lumbar puncture yielded 20 cubic centimetres of very slightly turbid fluid under pressure that was slightly less than previously.

On October 15, 1933, the temperature was 37·1° C. (98·8° F.). The patient was comfortable and his mentality was improved. Lumbar puncture yielded 12 cubic centimetres of very slightly turbid fluid under moderately increased pressure.

On October 16, 1933, an intramuscular injection of 60 cubic centimetres and an intravenous injection of 30 cubic centimetres of antistreptococcal serum were given. The patient was comfortable. Lumbar puncture yielded 20 cubic centimetres of very slightly turbid cerebro-spinal fluid, and 10 cubic centimetres of a 10% solution of urotropin and 10 cubic centimetres of air were injected intrathecally. In a cerebro-spinal fluid smear polymorphonuclear cells were moderately plentiful. No organisms were detected. Culture was sterile after forty-eight hours in incubation. An intravenous injection of 30 cubic centimetres and an intramuscular injection of 60 cubic centimetres of antistreptococcal serum were given.

On October 19, 1933, the patient was feeling comfortable. In the previous seven days the chart had been varying between 36·7° and 37·8° C. (98° and 100° F.). On lumbar puncture 15 cubic centimetres of clear cerebro-spinal fluid were removed under very slightly increased pressure.

ILLUSTRATIONS TO THE ARTICLE BY DR. J. V. DUHIG.

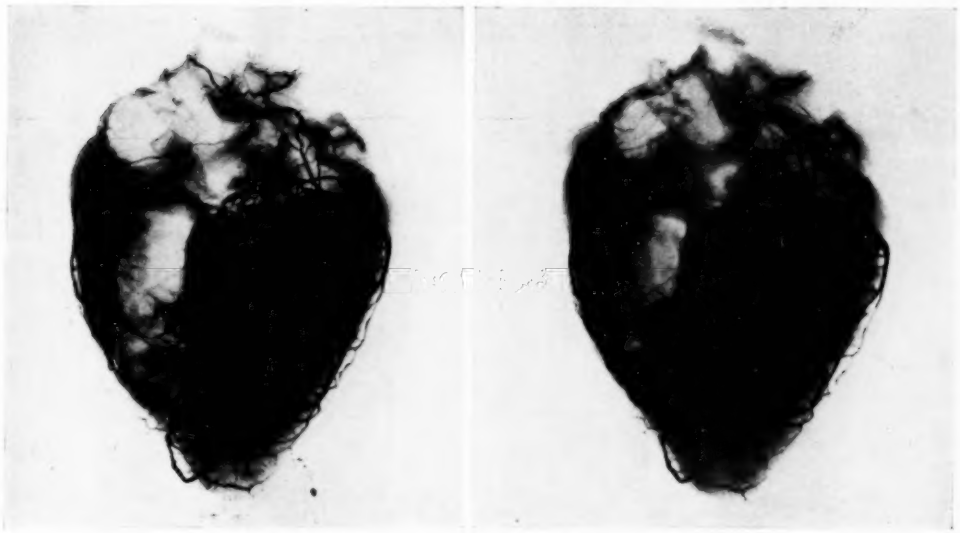

FIGURE I.

Stereoradiograph of the injected coronary arteries of a normal heart from a young woman accidentally killed.[1]

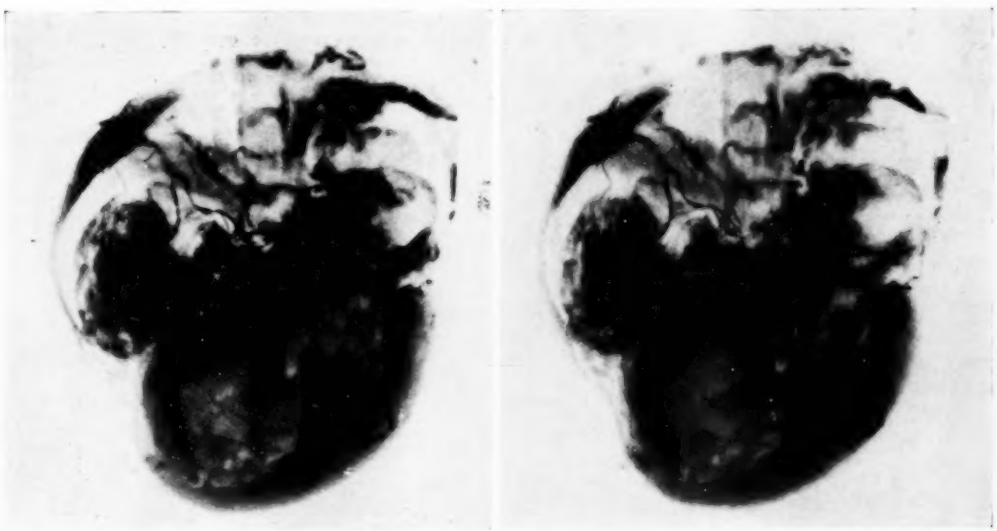

FIGURE II.

Almost complete bilateral coronary block from old man dying of rupture of the heart following infarction of the left ventricle. Note the numerous calcified areas at sites of miliary infarcts.

[1] Readers should cut out Figures I, II and III as they are and paste them on the usual stereophotographic mount and examine them with a stereoscope, when they will be seen in three dimensions.

ILLUSTRATIONS TO THE ARTICLE BY DR. J. V. DUHIG.

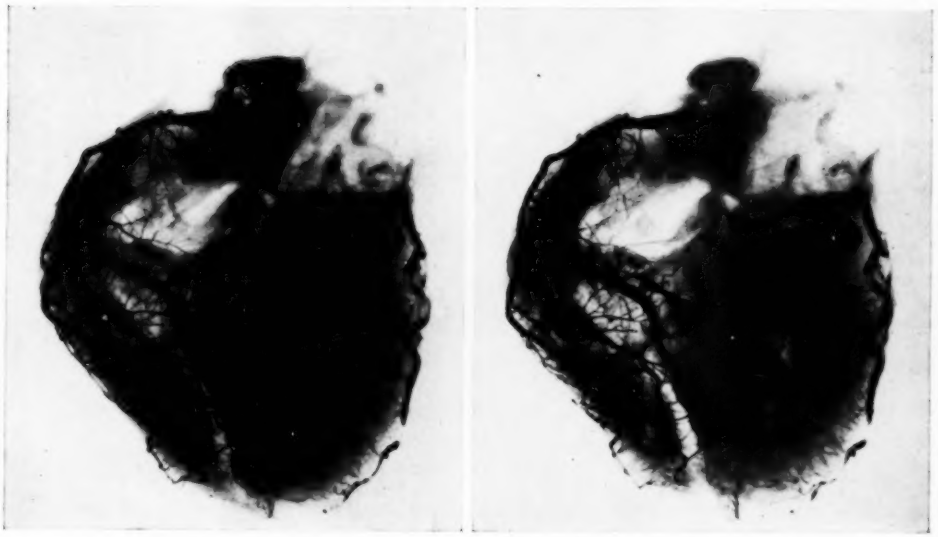

FIGURE III.
Partial left coronary occlusion with compensatory extension of the right side supply.

FIGURE IV.
Blood supply to septum of the heart after complete left-sided coronary block. Note absence of blood supply to right side of apex and to site of right side of bundle of His.

FIGURE V.
Capricious calcification of costal cartilage.

On October 24, 1933, lumbar puncture yielded 12 cubic centimetres of clear fluid under normal pressure. Culture was sterile. Seventy-six polymorphonuclear cells per cubic millimetre were found.

On November 7, 1933, the patient was allowed to get up.

On December 14, 1933, he was discharged cured. The posterior wound was healed.

Case II.

E.P., aged eleven years, was admitted to the Royal Alexandra Hospital for Children on October 29, 1933, complaining of left earache for five days with purulent discharge for one day. Pain and tenderness were present behind the left ear for two days. This morning she developed very severe generalized headache.

On examination the child was semi-conscious and crying out constantly, as if with meningeal irritation. There was a profuse purulent discharge from the left ear. The left mastoid region was slightly œdematous. Marked neck rigidity was present. Double Kernig's sign was present. The other systems were clear. On lumbar puncture 20 cubic centimetres of turbid cerebro-spinal fluid were removed under increased pressure. On examination of the fluid 710 milligrammes *per centum* of chlorides were found to be present; glucose was also present. Neutrophile pus cells were abundant. No organisms were found. Culture was sterile.

A left Schwartze operation was performed two hours after the patient's admission to hospital. Muco-pus was found in the mastoid antrum. The dura of the middle and posterior fossæ appeared healthy and was not incised.

A swabbing from the external auditory meatus and from muco-pus of the mastoid antrum revealed many Gram-negative intracellular diplococci.

A further 20 cubic centimetres of turbid cerebro-spinal fluid were later removed and 10 cubic centimetres of anti-meningococcal serum were given intrathecally and 10 cubic centimetres intravenously.

On October 30, 1933, lumbar puncture was performed and 20 cubic centimetres of cerebro-spinal fluid were removed; the fluid contained many pus cells, but no organisms. Antimeningococcal serum was given intrathecally, intravenously and intramuscularly.

On October 31, 1933, on lumbar puncture 20 cubic centimetres of cerebro-spinal fluid were removed; the fluid contained a few intracellular diplococci and grew meningococci on culture.

On November 1, 1933, blood culture was performed and meningococci were grown.

Until November 4, 1933, daily lumbar punctures were performed and antimeningococcal serum was given intrathecally, intravenously and intramuscularly, the total amounts being intrathecally 70 cubic centimetres, intravenously 120 cubic centimetres, intramuscularly 80 cubic centimetres.

On November 5, 1933, the patient's general condition was satisfactory and the cerebro-spinal fluid was clear and sterile.

Convalescence was interrupted by a severe serum rash and later by *Bacillus coli communis* pyelitis. The patient was eventually discharged cured.

An interesting feature of this case is the fact that the early diagnosis of the meningococcal meningitis was made through the presence of meningococci in the discharge from the middle ear and mastoid antrum two days before the organisms could be demonstrated in the cerebro-spinal fluid.

Both cases illustrate the importance of bacteriological examination of the discharge in *otitis media* and mastoiditis.

Acknowledgements.

I am indebted to Professor H. R. Dew and Dr. R. S. Godsall for their advice on the treatment of the first patient and to Dr. Uren and Dr. Lyons, resident medical officers at the Royal Prince Alfred Hospital and the Royal Alexandra Hospital for Children respectively for their care in the post-operative treatment and for the compilation of the data of the treatment.

Reviews.

HEREDITY.

MANY books on heredity published in recent years have dealt with the valuable and extensive studies made by biologists on plants and the lower animals. "The Chances of Morbid Inheritance", edited by C. P. Blacker, is, however, a publication of more direct significance to the medical profession.[1] It might even be regarded as one of the most important compendiums on this subject in English for either the general practitioner or medical student, despite the fact that it contains a few rather unfortunate errors.

The volume has the special merit that it consists of a number of chapters each one written by a specialist in his subject. Unfortunately, as is frequently the case in symposia of this kind, the chapters differ considerably in quality.

The subjects chosen for discussion and the respective authors are as follows: "Hereditary Nervous Disorders", W. Russel Brain, M.D.; "The Inheritance of Epilepsy", by the same author; "The Inheritance of Mental Disorders", A. J. Lewis, M.D.; "The Inheritance of Mental Deficiency", H. Herd, M.B.; "Hereditary Diseases of the Eye", Sir Stewart Duke-Elder, M.D.; "Hereditary Diseases of the Ear", R. J. Cann, M.S.; "The Inheritance of Asthma and Other Allergic Diseases", G. W. Bray, M.B.; "Hereditary Diseases of the Blood", L. J. Witts, M.D.; "Hereditary Cardio-Vascular Diseases", M. Campbell, M.D.; "Hereditary Renal Diseases", A. A. Osman, F.R.C.P.; "Hereditary Diseases of the Skin", L. Forman, M.D.; "Hereditary Gastro-Intestinal Diseases", M. E. Shaw, M.D.; "Heredity in Cretinism, Simple and Toxic Goitre", H. Gardiner-Hill, M.D.; "Heredity in Diabetes Mellitus and Renal Glycosuria", R. D. Lawrence, M.D.; "Heredity and Tuberculosis", E. R. Boland, M.R.C.P.; "Hereditary Neoplastic Diseases", A. Piney, M.D.; "Congenital Abnormalities of the Skeleton", Professor H. A. Harris, M.D.; "Appendix, The Analysis of Pedigrees", Professor L. Hogben, D.Sc.

Particular interest is at present being shown in the matter of human heredity, resulting from the general interest aroused by the problem of human sterilization and the attitude of modern governments to that problem. Judging, too, from recent very vigorous discussions between members of the medical profession in Australia, the relative importance of heredity and environment is a subject still calculated to bring out a surprising amount of ignorance as to the facts and knowledge already discovered. The volume under review provides some excellent information, together with some curious lapses which show only too well that certain of the writers were not properly familiar with the subject of genetics. Possibly this is not surprising in view of the almost complete absence of this subject from the medical curriculum, although it might be assumed that in a work like the present one a special effort would be made to see that the writers understood completely the mechanism of inheritance.

It is difficult in such a wide field to single out chapters for mention. The subject of mental deficiency and other mental disorders has been excellently treated. Dr. Herd was a member of the Mental Deficiency Committee of the British Medical Association. The idea that mental deficiency is a clinical entity—a popular belief still amongst some who ought to know better—is clearly disposed of. The discussion regarding sterilization here is well balanced and unbiased. The section on cardio-vascular diseases is also extremely good.

The chapter on heredity and tuberculosis cannot be said to err through any special favouring of the heredity side. The author shows how the whole attitude to the relative

[1] "The Chances of Morbid Inheritance", edited by C. P. Blacker, M.A., M.D., M.R.C.P.; 1934. London: H. K. Lewis and Company, Limited. Demy 8vo., pp. 462, with illustrations. Price: 15s. net.

importance of nature and nurture is too often purely superficial bias. "So pontifical are the utterances of the rival factions, as one must reluctantly call them, that it is difficult to avoid using religious analogies in referring to them, and although the reasonable man does not consider that the two creeds are incompatible or mutually exclusive, their protagonists appear to find them so." Unfortunately the author himself seems inclined to sit too much on the fence, and only refers to the recent work of Verschuer and Diehl in a postscript. These authors found that in a series of over one hundred twins there is much greater regularity in both being affected with tuberculosis (notwithstanding their environments) when they arise from a single egg (monovular twins) than when they arise as the result of the simultaneous fertilization of two eggs (binovular twins). In the first case, of course, both twins have exactly the same "hereditary substance". There is little doubt indeed that heredity plays its part in tuberculosis, but not in the way it was once thought to play it.

The statement that infantile amaurotic idiocy is confined only to the Jews is not correct. The section on the diseases of the eye leaves out much that is known, and the bibliography to this chapter is somewhat weak. Part of the chapter on the congenital abnormalities of the skeleton is irrelevant. The defects of the book are, however, amply balanced by its good points and should be easily corrected in another edition. The general idea of the book is to supply an answer for the general practitioner to queries regarding the probability of a given disease or defect being inherited. It is quite evident, reading between the lines, that whilst in many cases action would be problematic in regard to a general population, the course to be taken is clearly evident so far as individuals themselves are concerned.

It only remains to add that there is a short bibliography to each chapter, there is an index, which might be better, and an excellent appendix by Professor Hogben on the application of statistical methods to heredity studies. There are numbers of pedigrees and illustrations, and altogether the symposium is an able effort to meet a pressing demand from those who have neither the time nor the situation in regard to literature to delve for information into original and scattered publications.

POISONING.

DEATHS by accidental, suicidal and homicidal poisoning are unduly frequent, and much incapacity is caused by working amongst various substances necessary in different industries. It is therefore inevitable that toxicology is becoming a special branch of medical science. Erich Leschke, who is Professor of Internal Medicine in the University of Berlin, has supplied a definite need in his "Clinical Toxicology".[1] Leschke points out that up to 80% of poisoning cases are not properly diagnosed, at least in the beginning. In England cases of suicidal poisoning more than trebled within the years 1920-1930. In a series of 250 cases of poisoning in Germany during 1925-1927 veronal and "Luminal" together accounted for 14·6% of the total, exceeding any other group except carbon monoxide. Leschke states that in educated circles veronal is the method of choice for self-destruction. Thirty-two pages are devoted to carbon monoxide poisoning, and it is a melancholy fact that in Germany in 1929, 2,541 out of 3,636 cases of suicidal poisoning were due to coal gas, in addition to cases resulting from accident or carelessness. In the United States of America more than 5,000 persons die every year from carbon monoxide poisoning. An amazing instance of attempted suicide on the part of a doctor's daughter is reported. She gave herself an intravenous injection of two mils of metallic mercury, weighing 27·2 grammes, and recovered. Unusual means of effecting murder mentioned are: thallium rat poison (in food), "Novasurol" (injected by a physician), sodium silicofluoride (in food), cantharidin (the fatal dose being about thirty milligrammes) and strophanthin. Aconite has been similarly employed, and veratrine has been used with homicidal intent. Cyanides are not infrequently so used, and massive doses were thus given to Rasputin in pastry. The text explains how he escaped owing to alcoholic gastritis and hydrochloric acid deficiency. One cannot withhold sympathy with the householder who had a solution of cyanide in a beer bottle. A burglar breaking in drank this, with a fatal issue, and the householder was convicted of culpable homicide. Fatalities are described following the taking of aspirin, pyramidon, "Atophan", nitrobenzene, "Percaine" and "Novocain". In potassium chlorate poisoning a blue-green discoloration of the lips, nose and forehead is mentioned. Mention is made of "ginger paralysis" in America due to tricresyl phosphate, and of malignant tumours of the bladder in chronic aniline poisoning; also lymphosarcoma occurring in workers in cobalt ore, which contains arsenic. We learn that the petrol habit gives manifestations resembling alcoholism, and that poisoning by manganese dioxide dust simulates *paralysis agitans*. As regards tests for drunkenness, the estimation of alcohol in the blood is described; but this leaves out of consideration the important aspect of tolerance. The vexed question of the connexion of alcoholism with hepatic cirrhosis is not elucidated. We read of plumbism arising as a late sequela of gunshot wounds, and are told of the treatment of mercury poisoning by animal or blood (which surely should be wood) charcoal. Lead paralysis has not yet been adequately explained.

The book is an absolute mine of information concerning all sorts of poisoning, including honey, first described by Xenophon in his Anabasis; also the flesh of some fishes and their poisonous stings. Ergotism was first described in the year A.D. 857, and attention is drawn to its association with vitamin *A* deficiency. Dermatitis due to various plants and processionary caterpillars receives attention. Very little has escaped consideration, industrial poisoning being especially dealt with. Altogether it is an admirable book. Some quaint English words appear in the translation, such as "embolies", "vaso-dilatoric", "uptake" (for intake), "pupillar", "cloni" and "thrombitic". "Etiology" does not look well, and "basophilia are" is surely wrong. The constitution of antipyrin given on page 337 is incorrect.

[1] "Clinical Toxicology: Modern Methods in the Diagnosis and Treatment of Poisoning", by E. Leschke, translated by C. P. Stewart, M.Sc., Ph.D., and O. Dorrer, Ph.D.; 1934. London: J. and A. Churchill. Demy 8vo., pp. 354, with illustrations. Price: 15s. net.

LECTURES ON OBSTETRICS.

THE practice of many lecturers in publishing their lectures in book form, irrespective of their merit, has little to recommend it, and "An Outline of Practical Obstetrics for Nurses", by R. S. S. Statham, is no exception to the custom.[1]

The first chapter, on anatomy, is plainly and simply worded and a pleasure to read. In the rest much fault can be found. For example, in antenatal care the author states: "They [the breasts] should be washed daily, especially the nipples, care being taken to remove crusts." To allow the crusts to form at all shows a complete absence of antenatal care. The idea of washing is to remove the fluid which oozes through the nipples, and so prevent any crust formation which, being removed, leaves a raw, tender surface. When this and other defects are corrected the book will serve the useful purpose its author intended.

[1] "An Outline of Practical Obstetrics for Nurses", by R. S. S. Statham, O.B.E., M.D., Ch.M., F.C.O.G.; 1933. Bristol: John Wright and Sons, Limited. Foolscap 8vo., pp. 139. Price: 2s. 6d. net.

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RHEUMATISM.

THE possibility of preventing rheumatism has been discussed on many occasions and in many medical centres. The extent of the pathological changes following in the wake of acute rheumatism calls for every possible effort to prevent its occurrence. It is difficult to form an estimate of the extent of rheumatic damage, but if cardiac affection alone of all its possible sequelæ be considered, the importance of pursuing the subject becomes obvious.

The difficulty of devising means for the prevention of rheumatism is due to our ignorance of its causation. As pointed out recently in these pages, the view is generally held that the condition is bacterial in origin. The streptococcus is incriminated, some observers holding that a specific streptococcus is responsible, and others supporting what is known as the multiple streptococcal theory. Although it is recognized that a definite infectivity may sometimes be traced, and though epidemic-like infection has been known to occur, all workers agree that other factors, such as predisposition, climate and general surroundings have to be taken into consideration. These factors were discussed in 1927 in a report of a committee appointed by the Medical Research Council of the Privy Council; an abstract of this report will be found in THE MEDICAL JOURNAL OF AUSTRALIA of May 7, 1927. This report was a comprehensive document and was based chiefly on the medical and social histories of 721 families containing at least one child who had been or was under treatment for acute rheumatism; it is as valuable today as it was in 1927. Even if we presume that a streptococcus or some combination of streptococci is ultimately responsible for the disease, the discovery of additional factors does not simplify matters very much. For example, if family incidence were found, this incidence might indicate some special tendency to or inheritance of the disease. On the other hand, it might be an indication of similarity of environment or of contagion. The compilers of the Medical Research Council report were unable to find any direct relationship between the incidence of rheumatism in children of the hospital class and the degree of poverty. At the same time, general hygienic surroundings appeared to afford some protection. We may conclude, therefore, that the full story of acute rheumatism will not be told until the streptococcal maze, immunological as well as cultural, has been made patent, and until more is known of the rôle of what at present are believed to be additional factors.

The bacteriological problem is exceedingly complex. The question to be decided, once the association between streptococci and acute rheumatism is admitted not to be one of chance, is whether the organisms themselves produce the lesions by developing the property of selective localization in particular tissues or whether they remain in a localized focus. Again, if they remain in a localized focus, they may produce the pathological changes characteristic of the disease either by their toxins circulating in the blood or by the production of an antigenic factor that gives rise to a specific allergic response. Any of these mechanisms is compatible with the view either of a

specific streptococcus being primarily responsible, or of a variety of types with certain common properties as causative agents. The possibility of the existence of a focus in which the organisms multiply makes one think at once of the tonsil as the infective focus. An enormous amount of work has been done on all the possibilities that have been mentioned, but all that can be said is that no satisfactory conclusion can be reached in regard to any of them. Medical investigators are still groping more or less in the dark and are no nearer to the solution of the problem than they were several decades ago.

If we turn to the additional factors, we are not much better off. Possibly too little attention has been paid to them. That an hereditary predisposition to the disease exists has been held by many, and much can be said for their contention. It is not reasonable to lay all the blame on the seed and none on the soil in which it is sown. In this direction more can be expected of the biological chemist. In several centres investigations into the non-bacterial factors of acute rheumatism have been made. So far Australia has done nothing in this direction. At the Hobart congress held in January of this year, the Section of Medicine, at the instigation of Dr. J. Kempson Maddox, decided to recommend to the Federal Council of the British Medical Association in Australia that a full and comprehensive inquiry into rheumatism, similar to inquiries that have been conducted in other countries, should be undertaken. The Federal Council is to meet at the end of August, and it is to be hoped that the members of the Council will not let the suggestion emanating from the Hobart congress lapse. An investigation similar to that made by the Medical Research Council could be organized; since the conditions of life in the Commonwealth are different from those in Great Britain, it is possible that extremely useful data might be gathered. The investigation would, we hope, not be confined only to the non-bacteriological factors. The main thing is to decide at once on a definite course of action; two or three years is little enough time for an investigation such as this would have to be.

Current Comment.

CARDIAC OVERWORK IN HYPERTHYREOIDISM.

THERE has been and still is a good deal of argument concerning the true cause of the well known cardiac lesions in hyperthyreoidism. On the one hand it is urged that the effect of the excessive thyreoid secretion on the heart muscle is specifically toxic, whether the secretion is in this case normal or not; on the other hand there is considerable evidence pointing to the sheer overwork of the heart as the cause of its surrender. In this connexion it must be pointed out that the augmentation of the general metabolism not only speeds up the rate of the heart, but also brings about an increased physico-chemical activity within every muscle cell in the organ. The problem of the effect of essential overwork on the heart is a very interesting one: it recalls the difference of opinion that is found amongst physicians today on the subject of strenuous exercise, particularly when undertaken by persons past their first youth. Some maintain that the heart is meant to work, and should be kept tuned up to a definitely vigorous life; others that its content of energy is not unlimited and should be conserved. As in most controversies it is likely that the middle road is the wisest to follow, but this question involves the consideration of usage and physical training, and would lead us away from the present topic. F. R. Menne, O. N. Jones and N. W. Jones have carried out an experimental study which sheds some light upon the whole question of the effect of abnormal work upon the myocardium.[1] They selected a number of healthy rabbits and divided them into four groups; in one the depressor nervous mechanism of the heart was destroyed, in another ephedrine or desiccated thyreoid gland was administered in addition; in the third group thyreoid substance alone was administered to the point of toxicity, and the fourth group was used as a control. In the animals operated on the carotid canals were opened and the depressor nerves sectioned, a part being removed, and the carotid arteries were carefully denuded. The pulse rates of all the animals were determined by electrocardiographic tracings taken from terminals placed under the skin to form the conventional second lead. Control observations were carried out on the rabbits before any surgical measures were taken, so as to accustom them to the unusual procedures. Certain difficulties were encountered. For instance, this animal has naturally a rapid heart rate, and this apparently varies considerably in different individuals. Also, there was reason to believe that the depressor nervous mechanism was not completely severed in all instances, for in some cases it was not found possible to increase the rate of the heart by operative procedures. But on the whole reasonably

[1] *Archives of Pathology*, March, 1934.

concordant results were obtained. It was found that following the artificial neurogenic increase in the heart rate, degenerative lesions could be demonstrated in the hearts of 80% of the animals: large doses of desiccated thyreoid produced similar changes in 90%. As might be expected, a combination of these methods produced more signal changes in the heart muscle. The nature of the lesions was a swelling of the muscle bundles, loss of the usual transverse striation, the formation of fat vacuoles, and invasion of the muscle bundles by histiocytes and fibroblasts. The photomicrographs reproduced with the article show well the degeneration of the heart muscle fibres, with cellular invasion, and in the more intense and prolonged reactions, a definite fibrosis. The results obtained in the experiments in which tachycardia was induced by the administration of thyreoid extract were comparable with those in which the depressor nerves were divided. The effect of ephedrine was not to augment the results, as might have been thought, by a possible raising of the pulse pressure, but rather to diminish them. There was little or no modification of the body weight in the animals subjected to operation, and thus it would appear that the effects observed were due solely to a mechanical increase in the heart rate. The animals studied were kept in close quarters, too great an expenditure of physical energy being thus prevented; the authors consider that the experimental conditions in this respect approximated to the study of human patients in bed.

In discussing the results of this experiment it must be remembered that the animal chosen for investigation is small and active, and has a naturally rapid pulse rate (averaging 200 per minute); its general metabolic rate of living is thus higher than that of the normal human animal. This might make it easier to produce conditions of circulatory strain, especially in laboratory animals. Also, we cannot say certainly that the only effect on the heart of dividing the depressor nerves is to augment the rate of the heart. In the light of recent knowledge it is possible that there is also some chemical or other metabolic action at work. However, these criticisms are not designed to invalidate the work, which has been carried out with attention to details and with proper control. It certainly seems to indicate that the abnormal speeding up of the heart in hyperthyreoidism alone is capable of causing destructive changes in the cardiac muscle. We know the heart to have a naturally wide margin of reserve power, but when this is persistently drawn upon, this credit will soon become a debit, particularly in persons who have topped the rise of life and who are on the steady downward slope that leads to the inevitable degeneration of age. It has been pointed out that the human heart, under usual conditions, works for nine hours a day and rests fifteen hours; a recollection of these figures should serve as a reminder of the serious results that will accrue when the hard-driven heart of the hyperthyreoid patient is continuously forced to work overtime.

PERIARTERITIS NODOSA.

THE condition known as *periarteritis nodosa*, first described by Kussmaul and Maier in 1816, is interesting and rare. It may be described shortly as being characterized by aneurysmal swellings and nodular formation of the small arteries and accompanied by destructive tissue changes. The arteries in the body are not all equally affected; the resulting symptoms therefore vary within wide limits and diagnosis is difficult, if not impossible. Many observers, including Weichselbaum, Schmorl and Benedict, thought that the condition was a syphilitic manifestation. Others who differed from this view drew attention to the rarity of *periarteritis nodosa* as compared with the frequency of the occurrence of syphilitic vascular lesions, and to the absence of a response to the Wassermann test associated with it. Dickson, whose important contribution was published as long ago as 1907, held that *periarteritis nodosa*, generally so called, included two conditions. One, *periarteritis nodosa*, he regarded as a true periarteritis due to syphilis; the other he called *poly-arteritis acuta nodosa*, and described in connexion with it small localized nodules on the smaller and medium sized arteries. In reporting a case of this condition, R. B. Haining and T. S. Kimball appear to miss the double classification of Dickson.[1] They describe their case as one of *polyarteritis nodosa* and state that Dickson preferred this term. Limitations of space will not permit a discussion either regarding nomenclature or pathology; the history of their case alone will be considered. The patient was a negro, aged forty-nine years. He was ill for nearly a year before he died without a correct diagnosis having been made. It is on the missed diagnosis that Haining and Kimball lay particular stress. The patient had mild fever, a few carious teeth and tenderness over the left spinal accessory nerve. Both blood and spinal fluid failed to react to the Wassermann test. The patient left hospital with a diagnosis of neuritis, radiculitis and pharyngitis. On his second admission to hospital he had been unconscious and had paresis of the right arm and leg. He left hospital again, against advice, after twelve consultant specialists had failed to agree on a diagnosis. He became comatose and died soon afterwards. At autopsy the condition was not suspected until blocks of tissue were examined; the kidneys and heart were chiefly affected. The most important symptoms of this condition are œdema of the legs, fever, pain in the legs, polyneuritis, hæmaturia, cardiac insufficiency, melæna, cerebral symptoms *et cetera*. When the more superficial arteries are affected diagnosis may be possible; in other obscure conditions *periarteritis nodosa* might be considered as a possibility. One case is on record in which the process came to a standstill, and in two instances remission followed the use of arsenical drugs.

[1] *The American Journal of Pathology*, May, 1934.

Abstracts from Current Medical Literature.

OPHTHALMOLOGY.

Medical Treatment of Chronic Glaucoma.

W. ZENTMAYER (*New York State Journal of Medicine*, December 15, 1933) points out that miotics, to be efficacious, must be properly administered. A strength sufficient to keep the pupil contracted must be employed and for this reason the dose must be gradually increased. Pilocarpine may first be used in the strength of 0·015 gramme to 30 cubic centimetres (one-quarter of a grain to an ounce), increased to 0·24 gramme to 30 cubic centimetres (four grains to an ounce), or eserine may be given at bedtime or during the night. Conjunctival irritation may be allayed by including cocaine in the solution, by using fresh solutions, by using weaker solutions in the day time than in the night time, and by the use of an astringent collyrium. Adrenaline and its substitutes are of value to increase the efficacy of miotics. Ergotamin (gynergen) given by the mouth is useful as an adjuvant or may be substituted temporarily for the local treatment when folliculosis develops. Massage is useful. Osmotic treatment—the intravenous injection of 150 to 200 cubic centimetres of a 10% salt solution—may be employed before operation to reduce excessively high tension. In the great majority of cases operation is sooner or later required.

Scleromalacia Perforans.

J. VAN DER HOEVE (*Archives of Ophthalmology*, January, 1934) describes four cases of degeneration of the sclera which he calls *scleromalacia perforans*. Three of these cases occurred in women suffering from chronic rheumatic polyarthritis, and the fourth occurred in a man of sixty-nine who was otherwise healthy. The first patient also had cataracts, dulness of the cornea and conjunctival hyperæmia. Small and large holes in the sclera of both eyes were seen. Some of the holes were covered with conjunctiva, but some were not, the uvea being left bare. The holes were situated between the limbus and the equator. In the second case hypopyon was present. In the male patient the perforation was present at the limbus in both eyes.

Retinal Detachment.

C. DEE SHAPLAND (*The British Journal of Ophthalmology*, January, 1934) presents a review of 425 cases of retinal detachment occurring in patients treated at the Royal London Ophthalmic Hospital during the years 1929 to 1933. In this series a retinal hole or holes were found in 324 cases, or 76·2%. The round hole is usually situated about two disk diameters behind the *ora serrata*. There were 115 examples of the round hole, 113 of disinsertion at the *ora serrata*, 105 of the arrow-head, 20 of the radial slit-like tear, and 19 of the irregular rent. The temporal half of the globe is the commonest site for a retinal hole; 79·6% of all tears occurred in this situation. The infero-nasal is the least common situation. The arrow-head and horseshoe holes were found in the upper half of the globe in 86·6%, and in the supero-temporal quadrant in 60%. The disinsertion occurs mainly in emmetropic eyes and in the infero-temporal quadrant, a history of trauma being obtained in 39%. The hole need not necessarily be found on that portion of the retina which is detached at the time of examination. Starting from December, 1929, 221 patients were treated after Gonin's method, the cautery puncture, with the result that 27·6% were cured and the condition of 10·4% was improved. In 17 cases of "blind" operation, in which the hole was not found, no patient was cured. From February, 1932, the Guist-Lindner method of multiple trephining was used in 79 cases. Cures resulted in 25·3% and the condition of 15·2% was improved. In 22 "blind" cases 22·7% of patients were cured and the condition of 18·2% was improved. From August, 1932, to January, 1933, diathermy was used in 72 cases after the method of Larsson. In the Weiss machine the current was regulated to give a reading of 60 milliampères when it was passing through the patient. Of all the patients treated by diathermy 47·2% were cured and the condition of 13·9% was improved. When the retinal hole was found, the percentages were 57·7 and 15·0, and for the 12 "blind" cases 25% and 8·3% respectively. Larsson's operation is, in the author's opinion, the most satisfactory method of treatment.

Visual Sensation Produced by Röntgen and Radium Rays.

S. R. GIFFORD AND E. E. BARTH (*Archives of Ophthalmology*, January, 1934) recall a report by A. H. Pirie in 1932, entitled "Seeing with Closed Eyes". Pirie noted that when Röntgen rays were passed directly through the eyes without the interposition of a fluoroscopic screen, a sensation of light was produced, and that when a radio-opaque letter was interposed between the tube and the eye, it could be read. The effect was the same whether the eyes were open or closed. Pirie used the phenomenon in localizing foreign bodies in the eyes. Patients with dense opacities in the anterior segment, such as cataract, could see the letters if the retinæ were intact. It was always necessary previously to produce dark adaptation of from ten to fifteen minutes. No reference to previous work was made, but it was found that Röntgen referred to the subject in 1895, Edison referred to it in 1896, and Dorn and Brander in 1898. In 1902 Janal and Curie described the visibility of radium rays; but the most complete investigation was that by Himstedt and Nagel in 1901. Nothing more was written until R. B. Taft published a paper in 1932. The present authors have endeavoured to apply the phenomenon as a more accurate means of determining the retinal function than the usual tests for projection of light. It seems probable that the sensation produced is due to direct photochemical effect on the rods. It does not serve in judging of the macular function and in certain patients the results are unreliable.

Intracapsular Extraction in Highly Myopic Eyes.

A. ELSCHNIG (*Archives of Ophthalmology*, January, 1934) advises the intracapsular extraction of cataract in highly myopic eyes. The operation is usually an anxious performance for many operators. Homatropine is instilled, the eye is cocainized, and a retrobulbar injection of 2% procaine solution with epinephrine is given. A bridle suture is passed through the superior rectus muscle and a limbal section of two-fifths of the cornea is made with a conjunctival flap. A suture is then passed through the conjunctival flap and the root of the iris cut with de Wecker scissors. The capsule forceps is then introduced into the anterior chamber, the capsule being caught below the corner margin of the iris and the lens being tumbled by slow movements, while the squint hook presses on the lower margin of the cornea. An iridectomy may be performed if the pupil does not dilate well or if liquid vitreous escapes after the incision is made. Though the danger of vitreous loss is greater, the result is not worse than that for emmetropic or hypermetropic eyes.

OTO-RHINO-LARYNGOLOGY.

Tuberculosis of the Larynx.

DAVID I. TOBIN (*Archives of Otolaryngology*, February, 1934), in a paper dealing with tuberculosis of the larynx, concludes that tuberculosis of the larynx has not received the attention it deserves because of the prevalent idea among general practitioners that little or nothing can be done to relieve the condition. Routine laryngological examinations should be made in every case of pulmonary tuberculosis, since tuberculosis of the larynx is a common complication. Early diagnosis is essential to the successful treatment of this condition. In cases of chronic laryngitis which do not respond readily to removal of the ætiological factor, such as sinus infection or misuse of the voice, a careful study should be made to exclude a pulmonary focus. A disease of the larynx superimposed on a severe pulmonary tuberculous infection is tuberculosis of the larynx until proved otherwise. A larynx may heal while tuberculosis advances in the chest. The contrary rarely occurs. Patients with definitely diagnosed infections are best handled in a sanatorium for tuberculosis, where they are under constant observation.

Patients with early infections may improve with vocal rest alone. The actual electric cautery, used in cases in which infiltration and ulceration are present, will relieve symptoms and sometimes result in cure. Biopsy and other surgical procedures for absolute diagnosis should be employed as a last resort, since such a procedure may light up an otherwise quiescent focus.

Effects of Drugs on Ciliary Activity of the Mucosa of the Upper Respiratory Tract.

D. M. LIERLE AND P. M. MOORE (*Archives of Otolaryngology*, January, 1934), in a paper dealing with the effects of drugs on the ciliary activity of the mucosa of the upper respiratory tract, quote Yates, Hilding and Lucas as having pointed out that the activity of the cilia is an important physiological factor in the defence mechanism of the upper respiratory tract and that it is evident that any agent which interferes with the normal activity is detrimental to this defence mechanism. The authors conclude that tap and distilled water, when applied to the mucosa of the upper respiratory tract, cause slowing of the ciliary beat. A 3% solution of ephedrine hydrochloride is not detrimental to ciliary activity, but at times increases it slightly. A 5% solution of cocaine hydrochloride is not detrimental to ciliary activity, but 10% and 20% solutions produce definite slowing with good recovery. Mild silver protein in concentrations of 5%, 10% and 20% produces an initial speeding of ciliary activity. This is followed by a slowing, which may be due to the water solvent rather than to the drug. A 0·5% solution of eucalyptol has no deleterious effect on ciliary activity. A 0·5% solution of menthol and, to a greater degree, a 1% solution have a mildly depressing effect on ciliary activity. A 1% solution of thymol, a 0·5% solution of thymol, and a 1% solution of eucalyptol, in the order named, are definitely detrimental to ciliary activity. A 1 in 1,000 solution of epinephrine hydrochloride, a 2% solution of zinc sulphate, and a 2% solution of mercurochrome, in the order named, are definitely detrimental to ciliary activity. A 0·5% solution of silver nitrate is immediately and fatally detrimental to ciliary activity. In no instance was it possible to start the cilia beating again after its application.

Carcinoma of the Œsophagus Treated by Radiation.

F. J. CLEMINSON AND J. P. MONKHOUSE (*Journal of Laryngology and Otology*, May, 1934) comment upon the treatment of carcinoma of the œsophagus by radiation. The material on which the study is based consists of all the cases of carcinoma of the œsophagus treated in the throat department of the Middlesex Hospital during the years 1925 to 1932. In all there are 89 cases, 79 in men and 10 in women. Postcricoid growths have been excluded. Preliminary biopsy was carried out in 72 cases, of which 70 proved to be squamous-celled and one a spheroidal-celled carcinoma, while one was a myeloma. The general mode of treatment was as follows: First, an œsophagoscopy was carried out for diagnosis, with removal of a fragment of the growth for section; next, the patient was examined by X rays to determine the length of the stricture, and for this examination the Trendelenburg position is essential, otherwise the lower end is undefined. From this information the dose of radon to be used and the length of the applicator were determined. Finally, at a second œsophagoscopy the radon was placed in position and left for seven days. In the earlier cases radium bromide was used, but later on radon took its place. The radon seeds were applied by means of a Souttar's tube. The general attitude was to avoid gastrostomy if possible. It was found that the majority of the patients were able to swallow up to the time of death, and a preliminary gastrostomy was done only for a special reason and was not found to lengthen life. It was possible to obtain a *post mortem* examination in twenty-eight cases, and the results are given in tabular form. These results of treatment are so disappointing that the authors are left wondering whether it is not actually harmful to treat these unfortunate people by the application of radon to the centre of the growth, and whether they might not live longer if their only treatment were a preliminary removal of all teeth, followed by a gastrostomy. The only sign of a "silver lining" is that the number of cases in the series is too small for the figures to have anything more than a suggestive value.

Acute Pneumococcal Pharyngitis.

R. G. HENDERSON (*The Lancet*, March 24, 1934) records eleven cases of acute pneumococcal pharyngitis. Nine of the patients were women, aged from twenty-six to sixty-five, two were men, aged thirty-nine and forty-eight years. All the patients were in good physical condition. In all cases the onset was rapid, attended with fever of 37·8° to 40·5° C. (100° to 105° F.) and shivering. Local symptoms were remarkably constant, namely, severe sore throat, dysphagia, and very tender cervical glands, which in most of the cases were definitely enlarged. In all there was intense inflammation and œdema of the pharynx with membranous involvement of tonsils, uvula, soft palate, and posterior pharyngeal wall. Macroscopically the exudate was very similar in appearance in each case. Those seen early showed a grey, slimy, glistening, adherent exudate. Soon, however, there appeared erosion of the mucous membrane with petechiæ, and the membranous exudate rapidly became dark and necrotic. Local tissue destruction and sloughing resulted, but there was no characteristic fœtor. In three of the patients respiratory embarrassment was sufficient to warrant tracheotomy. In ten of the eleven cases there was an associated pneumococcal septicæmia. Only one patient recovered; all the others died rapidly. The pneumococcus was found as the predominating organism in the throats of all the patients, and all the blood cultures taken showed the presence of an associated pneumococcal septicæmia. Serological examamination of the various strains of pneumococci showed that one patient was infected with Type I, four with Type III, and six with Group IV pneumococci.

Recent Advances in the Physiology of Hearing.

W. J. MCNALLY (*Archives of Otolaryngology*, February, 1934), in summarizing his article on hearing in fish and hearing in man, states that it has been clearly proved that some fish can hear. The higher forms of fish are able to hear and differentiate between a number of tones and noises. The *pars inferior*, consisting of the saccule and the lagena, is the part of the ear most likely concerned with hearing in fish. The saccule has been shown to have no vestibular function in fish, frogs and a mammal, the rabbit. In pigeons it may be concerned with compensatory rotatory movements of the eyes. In man the cochlea is an organ of hearing. The basilar membrane is the most likely resonating mechanism for resolving the complex sounds falling on the ear into their simple constituent harmonic components. The apical part of the cochlea is concerned with the perception of tones of low frequency and the basilar part with the perception of tones of high frequency. The resonance theory of hearing or some slight modification of it is most likely correct. It is probable that simple nerve impulses are sent to the brain from each sector of the basilar membrane undergoing sympathetic vibration. A fusion of synthesis of these impulses takes place in the acoustic analyser, the nucleus of which is situated in the temporal lobe, the remnants of the analyses being distributed throughout the whole mass of the cortex. Bone conduction as generally tested may be misleading, owing to the fact that the noise present in the average room raises the threshold for perception of sound in a normal ear. Tests of bone conduction should therefore be conducted in a sound-proof room, or else the ear of the observer and of the patient should be closed in order to shut out the screening effect of the noise equally from both; in other words, the absolute bone conduction test should be used in addition to the usual tests. Lesions of the middle ear in early childhood cause a loss of hearing by air conduction of the higher tones rather than the lower tones. Tests of the hearing are of little value in detecting early otosclerosis, because involvement of the mechanism of hearing may be a relatively late phase of the disease. A metabolic test must be discovered to detect the disease in its very early stages.

Special Articles on Treatment.

(Contributed by request.)

XXXVI.

THE TREATMENT OF ACUTE TONSILLITIS.

ACUTE non-specific inflammatory diseases of the tonsil have been classified into various types according to whether the brunt of the inflammation affects the surface epithelium, the parenchyma, the follicles, or the peritonsillar tissues. The first three types are really different stages of the same disease merging one into the other. They vary only according to the extent and degree of the inflammation present. The fourth may be due to an extension of an acute lacunar infection into the peritonsillar tissues, but is more often the result of cryptic retention due to adhesions or fibrous hypertrophy blocking the tonsillar fossa or crypts. The point of chief importance to remember is that in all types of tonsillar inflammation there is a lesion of the epithelium and that some form of pathogenic bacteria has penetrated it and invaded the tonsil. Treatment therefore depends not so much on the type as on the virulence of the invading organism and on the patient's general condition.

There is no hard and fast line of treatment; each case needs individual consideration according to its requirements. It must also not be forgotten that an acute tonsillitis may usher in a specific fever; therefore careful examination should always be made of the body and limbs for rash. The simple catarrhal type is rarely an independent disease, but part of a general catarrhal inflammation of the upper resepiratory tract, such as we see in an acute cold. We are seldom called upon to treat this type, patients usually relying on home remedies.

Many are not ill enough to be confined to bed or even kept indoors, but in children, in whom this condition is commonest—probably due to increase of lymphoid tissue at this age—there may be a sharp rise of temperature, painful swallowing, and pains radiating to the ears, suggesting *otitis media*. Pain in the ear, however, is not necessarily the result of a middle ear involvement, but may be a reflex from the throat. If earache is complained of, particularly in children, the ears should be examined, as the inflammation may extend to the Eustachian tube. Attacks of pyrexia are common in children in this condition. In feverish attacks in children the throat and ears should always be examined, as children frequently do not complain of sore throat or earache. There may be some sneezing, cough, and alteration of the voice due to the general inflammation of the pharynx *et cetera*. Some tenderness of the tonsillar glands is usually present. An examination reveals some swelling and redness of the tonsils, but no visible deposits in the crypts.

If the attack is severe, the patient should be confined to bed in a room free from draughts, and of even temperature. The diet should consist of milk foods, custards, jellies and home-made lemonade. Commence treatment with a purgative, preferably calomel followed by a saline. For adults 0·015 gramme (one-quarter of a grain) doses of calomel are given at intervals of an hour for five or six doses; for children calomel is given in doses of 0·006 gramme (one-tenth of a grain) every hour for five or ten doses, according to age. Small repeated doses are preferable to a large single dose. If the temperature is high, a mixture containing *Spiritus Ætheris Nitrosi* and *Liquor Ammonii Acetatis* is useful in reducing temperature. Should the throat be dry, tincture of aconite in minim doses every hour for a few doses stimulates secretion and reduces temperature. If there is a rheumatic tendency, salicylate of soda, 0·3 to 0·6 gramme (five to ten grains) every three or four hours, or aspirin, 0·3 to 0·6 gramme (five to ten grains) every three hours, gives satisfactory results in reducing temperature and relieving pain. Phenacetin may be combined with the aspirin if the pain is severe. Alkaline sprays to the throat are better than gargles. Oily sprays containing menthol, 0·6 gramme to 30·0 cubic centimetres (ten grains to the ounce), or lozenges containing menthol, 3·2 milligrammes (one-twentieth of a grain), soothe the throat. Drops for the nose, such as ephedrine 1% with camphor and menthol, 0·06 gramme (one grain) each to 30 cubic centimetres (one ounce) of liquid paraffin, relieve the nasal stuffiness. If there is an accompanying tracheitis or laryngitis, inhalations of compound tincture of benzoin are helpful. Very little else is indicated for this simple variety. Topical applications in adults of silver nitrate, 1·2 to 2·4 grammes to 30 cubic centimetres (twenty to forty grains to the ounce), if applied early, frequently abort the attack or limit its duration. Recurring attacks in children are frequently due to enlarged tonsils and adenoids. The tonsils and adenoids should be removed in an interval. In adults nasal obstruction and sinusitis should be looked for and corrected when found.

Follicular or Lacunar Tonsillitis.

The follicular or lacunar variety is as a rule unilateral at the commencement, but within a few hours the other tonsil becomes infected. At first there is engorgement of the blood vessels of the tonsil and inflammatory exudate occurs into the parenchyma and crypts, resulting in tonsillar enlargement. The tonsillar pillars are hyperæmic and the uvula may be œdematous. Yellowish-white masses project from the crypts and when coalescing spread at times like a membrane over the surface of the tonsil, giving the appearance of a false membrane, which may be mistaken for diphtheria. In diphtheria the membrane is tough and firmly adherent to the tonsil, and if it is removed, the surface of the tonsil bleeds. In tonsillitis this membrane is soft, is easily removed, and there is no bleeding. The possibility of diphtheria should never be overlooked, and if there is any doubt whatever, a swab should be taken for bacteriological examination. The onset of the illness is, as a rule, sudden. There is a shiver or rigor, the temperature rising to 37·8° to 39·4° C. (100° to 103° F.), the pulse is rapid, there is headache, and severe muscular pains occur, particularly in the back and limbs. The tongue is furred and the breath is foul. The patient has no appetite and constipation is the rule. The tonsillar glands will be found to be swollen and painful, and there is pain and soreness in the throat, and painful swallowing, and there may be severe pains radiating to the ears. A full feeling in the ears due to congestion of the Eustachian orifices is not uncommon. The speech is frequently thick. Pronounced swelling of the glands of the neck suggests scarlet fever.

If the patient is seen early, abortive treatment may be tried, the entire tonsil being gently swabbed with silver nitrate, 1·2 to 2·4 grammes to 30 cubic centimetres (twenty to forty grains to the ounce). The solution should be applied by means of a cotton-wound applicator, care being taken that there is no excess of solution on the swab, for should some trickle into the larynx, violent spasms will be produced. This treatment is highly recommended by some authorities. Early cases are undoubtedly benefited by this treatment, but it is rare for patients to come under observation so soon.

The patient should be confined to bed and preferably isolated, particularly during epidemics of the disease. The frequent occurrence of tonsillitis in households, schools, or institutions calls for an inspection of the sanitary arrangements and milk supply. As recommended in the simple type, a purgative should be given at the commencement. If the temperature is high, tincture of aconite in minim doses, repeated every half hour until free perspiration results, will be found useful in reducing temperature in children. Nothing gives such good results in reducing temperature and relieving pain as aspirin, 0·3 to 0·6 gramme (five to ten grains), given every three hours, or if there is a rheumatic tendency salicylate of soda, 0·3 to 1·0 gramme (five to fifteen grains), with double the quantity of bicarbonate of soda, every four hours. The aspirin should be given in powder form mixed with honey. It will greatly relieve painful swallowing and is much appreciated. It is inadvisable to use cocaine, as it causes congestion and swelling and the throat consequently

feels blocked up afterwards. If swallowing is painful, "Orthoform" should be insufflated shortly before a meal.

Pain on swallowing may also be relieved by applying pressure with the hands just in front of the tragus and behind the ascending ramus of the jaw on each side during the act of swallowing. This is best done by a nurse standing behind the patient and applying the pressure upwards and backwards at the moment of swallowing. Semi-solids are easier to swallow than liquids. Hot fomentations to the neck about the angles of the jaw are very comforting and should always be applied, particularly when the glands are tender. Pain on swallowing is due to some peritonsillar inflammation and myositis, consequently the muscular action involved in gargling is painful and injurious and does more harm than good. It is better to irrigate the throat with a warm alkaline and antiseptic lotion every three hours (sodium bicarbonate and sodium biborate, 1·2 grammes of each to 30 cubic centimetres of water, or 20 grains of each to a fluid ounce, to which 0·18 cubic centimetre or three minims of carbolic acid solution are added) or the well known preparation eusol. The irrigation should be done with a Higginson's syringe, at least a pint of lotion being used. With the patient sitting up and leaning forward over a basin, the stream of lotion is directed on to the throat. Heat relieves pain, therefore the lotion should be as warm as the patient can stand it. The fluid displaces the tenacious mucus, reduces congestion, and relieves the pain. When the patient can open the mouth without pain or discomfort the exudate can be removed from the tonsil by means of a cotton-wound applicator dipped in hydrogen peroxide. This removal opens the crypts and by promoting drainage reduces the septic absorption.

If there is difficulty in opening the mouth, the peroxide can be used in the form of a spray. Various drugs, such as quinine, guaiacum, salol, sodium benzoate, and *Liquor Ferri Perchloridi* have been recommended. The last named, combined with potassium chlorate, is highly recommended by some. *Liquor Ferri Perchloridi*, 0·3 to 0·36 cubic centimetre (five to six minims) in glycerine, given every four hours, is a favourite and useful remedy in children. Painting the throat with 1% carbolic acid in glycerine or with equal parts of guaiacol in olive oil sometimes helps to check the inflammation. Oily sprays and lozenges, as previously mentioned for the simple variety, are useful in soothing the throat. Nasal operations should not be done when the tonsils are found to be unhealthy until such time as the tonsils are removed, as the operation is frequently followed by a severe tonsillitis. In severe epidemic forms and those secondary to nasal operation, anti-streptococcal serum should be given. The mouth should receive particular and constant attention from the outset. The course of the disease is usually favourable and terminates in from five to seven days, leaving the patient somewhat weak and exhausted, for which a tonic, such as *Syrupus Glycerophosphatum Compositus* (A.P.F.) or iron and arsenic may be prescribed. In recurrent cases complete removal of the tonsils should be done during a quiescent period. This affords complete relief from subsequent attacks with all their possible complications and sequelæ. Occasionally, after complete removal, lymphoid nodules occur on the posterior pharyngeal wall and lateral folds. These may become infected, causing symptoms similar to lacunar tonsillitis, but not so severe. The condition rapidly subsides if the nodules are painted with silver nitrate, as previously recommended for tonsillitis. A watch should be kept for rheumatism, endocarditis, albuminuria, and paralysis. If the last mentioned occurs, the case has almost certainly been one of diphtheria.

Abscess of the Tonsil.

An abscess sometimes forms in the body of the tonsil following an acute septic tonsillitis. The condition is rare and practically always occurs in adults. One tonsil is usually affected. The tonsil is swollen and inflamed, particularly over the site of the abscess. The surrounding tissues are not greatly involved. The patient complains of a painful swelling on one side of the throat, and general symptoms suggest lacunar tonsillitis. It is sometimes very serious, the patient being very septic and the temperature high. The abscess should be incised and the patient treated on the lines recommended for lacunar tonsillitis. In severe cases the tonsil should be enucleated.

Peritonsillar Abscess: Quinsy.

A peritonsillar abscess is a collection of pus in the peritonsillar space. It is due to an extension of infection from the tonsil into the loose peritonsillar tissues. The most important predisposing factor is retention of septic material in the crypts, particularly those of the tonsillar fossa, as the result of adhesions or fibrous hypertrophy. It sometimes follows an acute follicular tonsillitis, but may occur primarily. When pus forms, it is confined between the capsule of the tonsil and the muscular wall of the *sinus tonsillaris*. The accumulating pus strips the capsule from its bed and dislocates the tonsil inwards. The capsule may be entirely stripped from its bed, except where it is firmly adherent to the superior constrictor muscle at the lower pole. This stripping process may be limited to various situations by inflammatory adhesions; the tonsil may thus be dislocated in several directions. When the abscess occurs in the superior and external portion of the peritonsillar space, the tonsil is dislocated inwards and downwards. This is by far the commonest situation, for it is here that the capsule adjoins the tonsillar fossa. Anterior and posterior abscesses may occur, although rarely. The former pushes the tonsil backwards and inwards, and the latter forwards and inwards. These directions of dislocation should be kept in mind when you come to consider incising a peritonsillar abscess.

This condition is rarely met with in children and the aged, but is not uncommon between the ages of fourteen and forty. The symptoms of this condition in its early stages are similar to those of acute follicular tonsillitis, but rapidly become greatly intensified until the patient is in absolute misery from pain and dysphagia. Patients rarely apply for treatment until there is great swelling and discomfort and then the typical picture of quinsy is presented. The patient looks anxious, worn and ill. Saliva dribbles from his mouth, which he can only partially open. His head is held rigid and inclined to the affected side, and when he wants to turn his head and neck he does so from the shoulders. There is great difficulty and pain in swallowing, the pain radiating to the ear on the affected side. His breath and teeth are foul. His speech is thick and muffled, and tenacious mucus clogs his throat. Examination of the throat is difficult owing to the inability of the patient to open the mouth wide enough. In the majority of cases there is marked redness, œdema of the palate and uvula, with swelling, which is most marked over the upper part of the anterior pillar, while the tonsil is dislocated downwards and inwards towards the middle line.

Even if early treatment is adopted, it is doubtful whether any measures, local or general, will abort the attack. The measures recommended for acute lacunar tonsillitis should be adopted. In the evening a hypodermic injection of morphine allays the pain and discomfort, enabling the patient to sleep. The only treatment that is of any real benefit is surgical evacuation of the pus. This should be done as early as possible. In the typical palatal type the incision should be made in the palate half way between the top of the anterior pillar and the base of the uvula. This is best done after injecting a few minims of "Adrocain" at the site of the incision. A sharp-pointed bistoury should be used, bound with adhesive plaster to within an inch of its point. The knife should be pushed upwards and outwards to the depth of about 2·5 centimetres (one inch) and withdrawn with its cutting edge downwards and inwards so as to enlarge the opening. If pus is not struck, sinus forceps should be inserted in different directions and opened widely so as to evacuate the pus freely. Great relief follows the evacuation of the pus and rapid recovery follows. The following day sinus forceps should be passed along the track to keep it open.

When the abscess is situated posterior or anterior to the tonsil, it should be opened at its most prominent point. Enucleation of the tonsil has been recommended, as it evacuates the pus and removes the tonsil at the same sitting, but I do not recommend this procedure. After an

attack of quinsy the tonsils should always be removed, as the patient is liable to get recurrence, and at any rate such tonsils are unhealthy. Hypertrophied tonsils, especially those with lacunar exudate, strongly predispose the patient to lacunar tonsillitis or abscess, so such tonsils should be removed to safeguard the patient from these conditions.

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British Medical Association News.

SCIENTIFIC.

A MEETING OF THE QUEENSLAND BRANCH OF THE BRITISH MEDICAL ASSOCIATION was held at the B.M.A. Building, Adelaide Street, Brisbane, on May 4, 1934, DR. W. N. ROBERTSON in the chair.

The Cause of Death: A Review of Five Hundred Autopsies.

DR. J. V. DUHIG read a paper entitled: "The Cause of Death: A Review of Five Hundred Autopsies" (see page 112).

DR. ELLIS MURPHY congratulated Dr. Duhig on his paper; it was of very great interest to him and was the result of original observations. The statement that so many sudden deaths were due to arterial disease was not unexpected; in Dr. Murphy's experience as a physician arterial disease was the most common disease found on routine examination. Probably considerably more than 50% of patients who consulted him had changes in their arteries, and one would therefore expect to find it a common cause of death. Although many severe or fatal attacks of coronary thrombosis came on while the patient was at rest, it was not uncommon to encounter patients whose anginal symptoms dated from some definite severe effort and who later succumbed to a major attack of coronary thrombosis.

With regard to suicides, Dr. Murphy did not quite agree with the statement that all men were sane when they committed suicide. From his experiences with manic depressive patients he thought that most premeditated suicides occurred amongst such people or in border-line cases.

Dr. Murphy felt that Dr. Duhig's paper would do a lot of good, and he looked forward to reading it carefully in THE MEDICAL JOURNAL OF AUSTRALIA; it marked an epoch in pathological work in Queensland.

DR. G. W. MACARTNEY considered that many autopsies should be done to teach practitioners where they had failed in clinical diagnosis. He referred to the question of air embolus, in deaths resulting from abortions, and to Sir Bernard Spilsbury's views on this matter; also to the condition called *status thymo-lymphaticus*.

DR. W. H. STEELE inquired what were Dr. Duhig's grounds for assuming that all apical scars and pleural adhesions were tuberculous in nature.

DR. R. G. QUINN wished to congratulate Dr. Duhig on his paper and particularly on the excellent injections and photographs of coronary occlusion. He also wished to comment on the *Coroners' Act*. He agreed with Dr. Duhig regarding the undesirability of insisting on a *post mortem* examination and inquest in anæsthetic deaths. He drew attention to the uncertainty of a *post mortem* examination in the case of sudden deaths as illustrated by a case of suicidal cut throat, in which a *post mortem* examination was demanded, though viewing the body gave all relevant information, contrasted with the death of an infant that had been left for a couple of hours and had been discovered dead, the probable cause of death being asphyxia from aspirated food. Of the two cases, the second called for *post mortem* examination more than the first, though in the second case no *post mortem* examination was performed.

While agreeing with Dr. Duhig regarding the uncertainty of clinical diagnosis, Dr. Quinn wished to draw attention to the fact that even *post mortem* examination did not give complete certainty of diagnosis.

Dr. Quinn was interested to hear that Dr. Duhig regarded pyonephrosis as a very common condition in elderly patients, as in *post mortem* examinations conducted at the Brisbane Hospital before a pathologist undertook that work, there were seen many such cases; in one instance the diagnosis had been sarcoma of the kidney.

Dr. Quinn said that a salutary lesson to one's clinical observations was provided by a *post mortem* examination.

DR. J. LOCKHART GIBSON thanked Dr. Duhig for his valuable paper. His cases of calcareous disease of the coronary arteries reminded him of a clinical and pathological lesson he had received about forty years previously from the late Joseph Bancroft, who was a remarkable observer. A well known Brisbane man, who had been able to increase his life assurance to £3,000 three months before, died suddenly a few hours after returning to his home because of some epigastric pain. Dr. Bancroft took charge at the *post mortem* examination in a private house. He went straight for the coronary arteries. There was no atheroma of the aorta and no narrowing of the mouths of the coronary arteries, but on opening them up they were found to be narrowed calcareous tubes and containing thrombi. A few years later Dr. Bancroft himself died in much the same way while sitting in his chair. His relatives asked Dr. Gibson to do a *post mortem* examination. Dr. Gibson believed that he would not have found the cause of death except for Dr. Bancroft's lesson. Dr. Bancroft's arteries were narrow calcareous tubes after their first half inch or so. The aorta had no atheroma. He was only fifty-eight years old, though called "old Bancroft". Each of these men had been subject to occasional peculiar and extreme pallor. Dr. Gibson wondered if X ray screening might help *ante mortem* diagnosis, as it did so efficiently in cases of aneurysm of the third part of the aortic arch.

DR. ALEX. MURPHY joined his congratulations to those of the other speakers. One thing had struck him, and that was the number of deaths from coronary disease that occurred while the patient was asleep or at rest, and Dr. Murphy had found that this seemed rational. The demand for blood was less, the circulation slower, and therefore thrombosis was more likely to take place. It was an interesting fact that some autopsies gave no information. At the London Hospital statistics showed that about 12% of autopsies failed to reveal any clue as to the cause of death.

Dr. Duhig, in reply, said that he was very happy to accept Dr. Ellis Murphy's correction about the state of mind of suicides. He merely accepted the history he got at the time, and in all the men but two, careful inquiry had failed to reveal anything. Of course, as Dr. Murphy suggested, if one went back over the man's history very carefully, one might find that some were border-line cases. Suicides formed a very large percentage of these sudden deaths, and it was a severe stigma on the family. In future he would make more careful inquiries, if possible.

Replying to Dr. Macartney, Dr. Duhig said he had never seen a case of air embolism. He had read Spilsbury's paper very carefully, and had seen only one abortion at autopsy which was not septic, and in that case a pulmonary embolus was present. With regard to the deaths from *status thymo-lymphaticus*, the report of the committee on the condition, published by Turnbull, went to show that there was no entity "*status thymo-lymphaticus*" and that it was not responsible for sudden deaths. But still able clinicians and pathologists said that there was a similar condition which tended to sudden death.

In reply to Dr. Steele, Dr. Duhig remarked that apical scarring and to a certain extent pleural adhesions were accepted as presumptive evidence of pulmonary tuberculosis. Quite a number of apical scars were caseous and were very obviously tuberculous.

In reply to Dr. Quinn, Dr. Duhig said he did not agree that mere inspection of a suicide was enough, and he always made a thorough examination. Possibly there was some stigma attached, and if one could remove this

and help the family one should do so. It was never his intention to create the impression that clinical medicine was slipshod. He said, as a result of fairly extensive experience, that clinical medicine must be extremely difficult; present knowledge and methods were quite useless on occasions in the diagnosis of conditions found *post mortem*.

A MEETING OF THE SECTION OF MEDICINE OF THE NEW SOUTH WALES BRANCH OF THE BRITISH MEDICAL ASSOCIATION was held at the British Medical Association House, 137, Macquarie Street, Sydney, on June 7, 1934, DR. S. A. SMITH, the Chairman of the Section, in the chair.

The Treatment of Emergencies in Cardiac Disease.

DR. M. C. LIDWILL read a paper entitled: "The Treatment of Emergencies in Cardiac Disease" (see page 108).

In asking for a discussion on Dr. Lidwill's paper, the Chairman, Dr. S. A. Smith, remarked that in accordance with the advice given in a recent leading article in THE MEDICAL JOURNAL OF AUSTRALIA, he did not propose to call on any member individually. He expressed the hope that any member who had something to say would say it, and, also in accordance with the suggestions in the aforementioned leading article, would "make it snappy".

DR. WILFRED EVANS said that there were certain aspects of Dr. Lidwill's paper that he should like to comment on. First, in regard to venesection. This was an excellent procedure in any condition of cyanosis and congestion due to heart failure. Dr. Evans did not, however, see eye to eye with Dr. Lidwill in the matter of technique. Dr. Evans was in the habit of using a needle himself; but it was necessary to use a needle of a suitable type. He showed a needle that he had obtained in Vienna; it was broader at the bevelled tip and narrower at the outlet. Dr. Evans remarked that there was no clotting when this needle was used. Dr. Evans also pointed out that venesection was not only of value in reducing the congestion of the right side of the heart; it also reduced the viscosity of the blood and rendered the heart's action easier.

Dr. Evans went on to say that he was in agreement with Dr. Lidwill concerning the value of diuretics, such as "Salyrgan" and ammonium chloride. Dr. Lidwill had not mentioned urea, which was very useful. Dr. Evans often found it valuable to give "Theocin" and urea on the intervening days between the days on which "Salyrgan" was injected. He pointed out that "Theocin" was not so effective if given continuously, and a better result was obtained if it were given every fourth day.

Dr. Evans remarked that his experience with regard to trinitrin had been similar to Dr. Lidwill's. He had found that the tablets of trinitrin were of greater value than amyl nitrite. He pointed out that in *angina pectoris* trinitrin was of more use than morphine, as morphine took half an hour to act. He mentioned a case in which trinitrin had been invariably efficacious in the relief of *angina pectoris*.

Dr. Evans thought that tapping the pericardial sac was very seldom required. There was a large danger from the procedure, and it should be done only when the heart was gravely embarrassed. When the pressure on the heart was relieved by the removal of fluid the heart was apt to dilate and death might ensue. He had usually found that the ordinary measures of treatment with diuretics *et cetera* had been adequate. He thought that if fluid had to be removed from the pericardial sac, half of the amount removed should be replaced by air, so that there would not be too great an alteration in the pressure.

Dr. Evans thought that the administration of glucose in cardiac failure was a valuable measure; but he doubted the efficacy of insulin in such a small dose as five units three times a day. If the patient's pancreas was secreting insulin he could not see the object of giving a little more.

Dr. Evans expressed the opinion that quinidine had its most useful effect in the treatment of auricular fibrillation in thyreotoxicosis. The patient should always be given quinidine on the fourth day after operation for toxic goitre. An examination of two series of statistics showed that this method of giving the drug was almost invariably successful; but success was not so likely to attend the administration of the drug if it was delayed till three or four weeks after operation.

Dr. Evans thought that Dr. Lidwill might with advantage have stressed the importance of rest a little more. In his experience, if a patient was not given prolonged rest after a cardiac emergency, in a little time there was another cardiac emergency. As far as Dr. Evans could see, the prevention of cardiac emergencies in many of these cases would entail keeping the patients in hospital permanently —a counsel of perfection.

DR. JOSEPH COEN expressed his appreciation of Dr. Lidwill's paper and remarked that a pleasing feature was that the paper was the outcome of Dr. Lidwill's practical experience. The recording of the results of experience in these matters was very valuable, as the desired objects were not always achieved by the use of ordinary therapeutic procedures.

Dr. Coen agreed with Dr. Lidwill in the matter of venesection, and he used a similar method. He remarked that the quicker the relief to the right ventricle by venesection, the quicker would be the recovery of the left ventricle.

He thought the administration of insulin and glucose was a valuable measure. He was accustomed to giving 30 grammes of glucose morning and evening. He thought insulin helped to metabolize the glucose. But he pointed out that the method was still on trial, and it was only by physicians relating their experiences of it that a decision as to its value would be made possible.

Dr. Coen remarked that in the treatment of cardiac œdema due to cardiac failure the physician's therapeutic ability was tested to its utmost. The rational method was the administration of digitalis; but sometimes digitalis did not seem to have much effect, even if given in large doses, until some of the fluid was removed. The effects of "Diuretin" and "Novasurol" were at times dramatic; but the administration of "Novasurol" should not be frequently repeated. If the action of these drugs was putting out of action the cells of the renal tubules, any nephritis that might be present would be likely to be made worse. Dr. Coen thought that the continued use of these drugs was perhaps dangerous; but he remarked that any method ridding the patient of fluid would be a help to the subsequent measures of treatment.

Dr. Coen went on to say that the use of quinidine should be restricted to the treatment of certain conditions. The administration of quinidine in paroxysmal tachycardia had not been successful in his experience. If there was no decompensation, it might be of value in other conditions. Dr. Lidwill had mentioned the danger in an increased ventricular rate. Dr. Coen was not in agreement with him concerning the cause of the increased rate, namely, an increased conductivity of the atrio-ventricular node. Dr. Coen was of the opinion that quinidine depressed the conductivity, and the reason why the rate increased was that with the slowing of the auricular rate due to quinidine, the tendency to "block" at the atrio-ventricular node, which was induced in auricular fibrillation, was diminished and the ventricular rate was apt to take up the slowed auricular rate. Quinidine did not increase the conductivity of the atrio-ventricular node; it depressed the rate of conduction.

Dr. Coen pointed out also that quinidine depressed the vagus, which effect was likely to quicken the ventricular rate.

In the treatment of coronary thrombosis Dr. Coen remarked that morphine was best. He pointed out that in aiming at a sufficient dosage the medical practitioner must be careful to avoid overdosage. He had recently seen a man whose life had been endangered by overdosage with morphine; he had developed Cheyne-Stokes respiration. The shallow respirations induced by too much morphine were themselves a source of danger. Only sufficient morphine should be given to provide rest for the patient and to keep the pain in subjection. Dr. Coen preferred to remain inactive in regard to other measures of treatment for coronary thrombosis. He pointed out that the various experiments with drugs as coronary dilators had been made on healthy hearts, and went

on to say on to say that even if the coronary vessels were dilated it was not known that the coronary circulation would be increased in efficiency. Drugs used for dilatation of the coronary vessels would be apt to cause a fall in the blood pressure; therefore it was reasonable to suggest that the blood flow would not be increased. He did not think the use of these drugs was contraindicated; but he was sceptical about their value. If they were capable of increasing the flow of blood through the coronary vessels, it was logical to use them. Another possible disadvantage of vaso-dilators was that they might quicken the heart rate; he thought this was dangerous in coronary thrombosis. He pointed out that one could not know how close to death from cardiac rupture a patient with coronary thrombosis might be.

Dr. Coen concluded by again expressing his thanks to Dr. Lidwill.

DR. A. S. WALKER said that he felt he had to be cautious in rising to speak at a meeting after having read the leading article to which the Chairman had referred; but if his friends thought it desirable, they could amuse themselves by fitting him in the category that suited him best.

Dr. Walker remarked that Dr. Lidwill's paper was useful and practical.

He referred to the vague condition known as cardiac asthma, and mentioned that two patients recently attended by him had seemed to be benefited by the administration of "Coramine".

Dr. Lidwill had also spoken of pulmonary œdema. Dr. Walker confessed to being more than a good deal mystified by pulmonary œdema. In some cases he believed it was a manifestation of coronary occlusion. He mentioned that he had recently been called to see a patient who presented an amazing spectacle. She was the fattest woman he had ever seen; she was blue and gasping, and was expectorating pints of serous, frothy, pink liquid. He had thought that this patient would surely die; but he had given her atropine in large doses, and morphine, and she had made a good recovery. The ætiology in this case was doubtful, but the patient's configuration suggested fatty infiltration of the heart. In Dr. Walker's experience some of the patients most seriously afflicted with pulmonary œdema were arteriosclerotic and glycosuric.

Coronary occlusion was truly a medical emergency, and one that had to be treated with the utmost respect. The patient must be moved as little as possible, even being put to bed without being undressed. Dr. Walker had had a couple of experiences in which a little indiscretion in the way of movement had apparently caused death. One man had walked down stairs and up again and down again to a car after having been advised to go to hospital, and had walked into the hospital from the car and had shortly afterwards died.

Dr. Walker said that he should like to hear an expression of opinion on "Lacarnol", which Dr. Lidwill had merely mentioned in his paper.

In regard to venesection, Dr. Walker remarked that all recognized the necessity of the measure; but the patient might be difficult to bleed. It was almost impossible to get blood to flow through a small needle. Dr. Mills's method was merely to slip a scalpel into the vein. This was the method of their forefathers, who probably had been much more expert in venesection than medical practitioners of the present day.

Dr. Walker then referred to the administration of glucose in cardiac failure. Insulin was necessary if there was glycosuria. The administration of glucose seemed logical and sometimes he thought that patients were better, but he did not know. He recognized the justice of Dr. Evans's remarks concerning insulin: if the patient's insulin secretion was good, why add more? Dr. Walker thought that Dr. Lidwill's dose of glucose was very modest. He thought that much more could be given, provided the patient was not nauseated and did not vomit. This was a very important point; persons with congestive heart failure were easily nauseated.

Dr. Walker said that he had never tapped the pericardial sac. He thought that in the cases seen by him there had been no necessity for the measure. If he found that it was necessary, he would take care to remove little fluid; but he hardly thought he would undertake to produce the bubbly condition of the pericardial fluid by the means suggested by Dr. Evans.

DR. LAURENCE HUGHES referred to the use of glucose in cardiac conditions. He had had little experience of its use in adults in this respect, but he advised it in most cases of diphtheria, particularly of the toxic faucial type. The frequently associated hypoglycæmia in this condition seemed to be a sufficient rationale for the use of glucose. In severe cases insulin might be given in conjunction with the glucose, providing a check on the blood sugar content was available.

Dr. Hughes also referred to a case of acute pulmonary œdema; the patient was a woman in the ninth month of her pregnancy. The condition had responded well to morphine and atropine, and a normal labour had ensued. This had been twelve years previously and the patient had never had another attack, although she had been under observation and treatment for hyperpiesia at intervals ever since. Dr. Hughes said he would like to have the opinion of other members as to the prognosis in acute pulmonary œdema.

DR. KEMPSON MADDOX said that the subject had been so well covered by Dr. Lidwill and other speakers that it was difficult to add anything to the discussion.

Dr. Hughes had asked for information concerning the prognosis in cardiac asthma. Dr. Maddox's own experience was not sufficient to call on; but he was able to quote from the most recent "Medical Annual", in which it was stated that the average expectation of life in cardiac asthma and pulmonary œdema (said to be different grades of the same condition) was about eighteen months.

In regard to the value of insulin and glucose, Dr. Maddox said that for the past two years he had alternately given glucose alone and glucose with insulin to patients suffering from various types of congestive heart failure and coronary thrombosis. He had been able to find no difference in the results in his few cases. Shirley Smith's advice was, if there was no improvement after four days, to double the quantity of glucose and insulin.

Dr. Maddox gave a tip concerning venesection, remarking that the blood seemed to clot even more readily than usual in these cases. If sterile sodium citrate solution was allowed to drop on the wound from a bottle and an ordinary ether dropper, clotting would not occur.

Dr. Walker had asked for information about "Lacarnol". In his experience, "Lacarnol" had appeared to give relief in several cases; in fact, some of his patients had given up trinitrin for it. The question of expense was a real one.

Concerning coronary occlusion, Dr. Maddox said that he would make one or two provocative remarks, which might seem heretical. It had been seriously suggested that patients with coronary occlusion should not be given morphine, but encouraged to move in bed, in order to improve the coronary circulation. Absolute rest might, as a result of the stasis, cause an increase in the size of the clot. The time of danger of cardiac rupture was not at the time of the occlusion, but six to ten days later. Even restlessness might be Nature's means of increasing the blood pressure and so helping to maintain a better collateral circulation. If the pain was due to an accumulation of metabolites in the heart muscle cells, glucose could to some extent relieve the angina. Vaso-dilators also came into the scheme of treatment. Even the use of adrenaline had been advocated. Dr. Maddox remarked that he would be afraid to make a start with this type of treatment. For humane reasons alone it was necessary to apply some measures, such as the administration of morphine, for the relief of pain.

In conclusion, he remarked on the great amount of practical information in Dr. Lidwill's paper.

DR. J. H. HALLIDAY said that there was said to be some risk of embolism occurring with the altered cardiac rhythm after the administration of quinidine. Clark Kennedy had remarked that two of a number of carefully selected patients who had been admitted to the London Hospital for quinidine treatment, had died of cerebral embolism within twenty-four hours of admission, before

treatment had been commenced. He had been interested in Dr. Maddox's remarks about movement in coronary occlusion. He had always been impressed by the fact that in *angina pectoris* the patient was afraid to move because the pain tended to increase, whereas in coronary occlusion there was great restlessness because the patient realized that, whatever he did, he could find no relief, the picture resembling that of an animal in mortal agony.

DR. C. G. McDONALD said that all would agree that Dr. Lidwill was a physician of very long experience and that he should be able to add to the knowledge of those present. It would also be agreed that, though he wrote seldom, what he did write always evoked comment. At the conclusion of his paper Dr. Lidwill had half congratulated himself on his brevity. Dr. McDonald had wished that he might in the interests of clarity have expanded a little more.

Dr. McDonald remarked that he was worried over Dr. Lidwill's description of the cardiac circulation. He presumed that the Thebesian vessels were the old *venæ cordis minimæ*. If they were veins, then he took it that the flow of blood in them was in the opposite direction to that in Dr. Lidwill's description of their function. Dr. McDonald had not heard before that there were actual communications between the coronary arteries and the chambers of the heart. He presumed this was a new discovery. He should like to ask Dr. Lidwill if the so-called Thebesian vessels were really arterial.

Dr. McDonald said that he was worried about the dosage of atropine proposed by Dr. Lidwill. He remembered giving one-thirtieth of a grain to a patient suffering from drenching night sweats due to pulmonary tuberculosis. Half an hour later he had been called back to the ward to find that the patient had leapt from the bed and was excitedly pointing to the ceiling, where he believed he could see "myriads of rats". On another occasion Dr. McDonald had given one-fortieth of a grain without effect and had repeated the dose at the end of an hour, with the result that atropine poisoning had occurred. He thought these doses were heroic. He asked Dr. Lidwill whether atropine in big doses had any effect on heart block and whether Dr. Lidwill had had any experience of it.

Dr. Lidwill had staggered Dr. McDonald by his reference to many proprietary medicines. He supposed that these medicines should be tried, otherwise there was a likelihood of a discontinuance of the steady supply of blotting pads that came from the various manufacturers. If nobody tried them, knowledge of proprietary drugs would never come, and a few of these drugs were certainly valuable. Dr. McDonald was particularly interested in "Digoxin". He understood that this drug had the advantage that it was chemically and not physiologically standardized.

He had been pleased to hear Dr. Lidwill speak of the value of venesection. He agreed that the measure was not used frequently enough. In hyperpiesia without renal or cardiac failure, when the patient complained of palpitation and a sense of fullness in the head, striking relief was obtained by venesection. It should not be done if there was anæmia.

In conclusion, Dr. McDonald remarked that all those present should be grateful to Dr. Lidwill for the entertainment he had given them. A large number of questions had been put, and he hoped that Dr. Lidwill would do his best to answer them all.

DR. E. H. STOKES, after expressing his thanks to Dr. Lidwill for his paper, said that Dr. Evans had brought out an important point in stressing the value of rest in cardiac failure.

In regard to venesection, Dr. Stokes remarked that it was important to have the tourniquet correctly applied, at the right pressure. He pointed out that the best type of tourniquet was one provided with a pad that could be applied with any desired amount of pressure directly over the vein. He had found Petit's tourniquet useful for this purpose. The needle used by Dr. Evans was similar to one used by Dr. Stokes in blood transfusion. Liquid paraffin applied to the skin was helpful in the prevention of clotting. In case of emergenecy an ordinary razor was very useful for making the incision. The double-bladed knife used by the older physicians was also a very handy instrument.

Dr. Stokes said that most people had spoken as though the administration of insulin was of no danger. He drew attention to a case that had been mentioned in a paper read by Dr. L. W. Dunlop before the section some time previously. The patient was suffering from arteriosclerosis and had been under Dr. Stokes's care in Sydney Hospital; when insulin had been given the patient had developed heart block, and when the administration of insulin had been suspended the heart resumed its normal rhythm and rate.

DR. B. W. STEVENSON said that two years previously he had had the pleasure of listening to Dr. Lidwill at a meeting of the Eastern Suburbs Medical Association. Since then he had used "Diuretin" extensively; he thanked Dr. Lidwill for the help he had given him by pointing out the advantages of "Diuretin".

Referring to acute cardiac conditions, Dr. Stevenson remarked that though Dr. Lidwill had mentioned the use of phenobarbitone in his earlier address, he had not done so that evening. Dr. Stevenson thought that in many of these conditions there was a vicious circle that had to be interrupted by the use of a nerve sedative.

DR. S. A. SMITH expressed his thanks and congratulations to Dr. Lidwill on having stirred up one of the best general discussions at a meeting of the Section of Medicine for some time.

Dr. Smith said that he realized that any opinions that one could express would be merely impressions; they were not based on the soundest of premises. Before an authoritative opinion could be given on the value of insulin and glucose, "Lacarnol", rest *et cetera*, a large series of cases compared with controls would have to be investigated. As it was, general impressions only were possible.

In discussing pulmonary œdema, Dr. Smith remarked that some years previously he had listened to a lecture by Professor Frazer, in which the pathological physiology of the condition had been discussed. Dr. Smith said that he had tried to follow and understand the speaker, but had failed, and he now made public confession to that effect. He had come to accept the view that acute pulmonary œdema was an expression of acute and sudden left ventricular failure, frequently a manifestation of coronary occlusion. The more he saw of the condition, the more did he feel that this was the best explanation. If it was correct, then treatment with morphine and atropine was the best. Dr. Smith asked Dr. Lidwill why he gave strychnine in pulmonary œdema, and whether he thought venesection was of any value.

Several speakers had discussed the value of insulin and glucose. Dr. Smith felt that at first sight it seemed ridiculous to give such small doses of insulin to a patient whose pancreas was apparently functioning normally. Nevertheless he had given larger doses of glucose than those mentioned, and insulin in a dose of five, seven or ten units, three times a day, and he thought the procedure was of some value. He admitted that he had had no control cases. He mentioned a patient with hypertensive heart disease and cardiac failure to whom he had given glucose and insulin without changing the treatment in any other way. There had been a gradual improvement and the patient had been able to get about; the patient had then left off the glucose and insulin and had become worse; later he had recommenced taking glucose and insulin and had again improved. Dr. Smith said that the improvement in this case might not have been due to the glucose and insulin, but the facts were suggestive.

Dr. Smith said that he had had no success in the treatment of heart block with atropine. He took the view that no treatment was required unless the rhythm was inconstant. He thought that many so-called Stokes-Adams attacks were due to ventricular asystole. Barium chloride had been valuable in his experience in the treatment of attacks. He had only known one case in which there had been discomfort from the ingestion of this drug.

He had found "Lacarnol" very disappointing and had not been able to convince himself that it had any value.

Dr. Smith said that there were quite a number of points in Dr. Lidwill's paper that he should like to discuss; but he would end with mentioning one of them.

He had been astounded to hear Dr. Lidwill say that the coronary circulation could be increased by 45% by the action of theophylline.

Dr. Lidwill in reply said, in regard to Dr. Smith's last remark, that he would go further and say that "Euphyllin" in heart-lung preparations increased the coronary circulation by 90%.

In reply to Dr. Evans he said that he had been glad to hear of the needle for venesection. The use of urea between injections of "Salyrgan" was something new to him. He did not agree with Dr. Evans in his views concerning the prolonged use of "Theocin". He sometimes gave "Theocin" for months. He mentioned that a patient of his with *angina pectoris* had taken "Theocin" for twelve or eighteen months; the patient had obtained very little benefit for some months, but had adjusted the dose to suit himself and had greatly improved. Dr. Lidwill often found that there was little result from treatment with "Theocin" under a couple of months. In regard to tapping the pericardium, he remarked that if a medical practitioner had a large number of children under his care he would be sure to have some who would be greatly distressed by pericardial effusion and for whom something had to be done.

In regard to the use of insulin, Dr. Lidwill said that he was of the same opinion as Dr. S. A. Smith. Insulin was dangerous in heart disease if it was given without glucose; when insulin had been first introduced there had been some tragedies because this fact had not been recognized. But if the patient was protected against the insulin with glucose, insulin was undoubtedly useful. He had been giving glucose for a long time, then had commenced giving insulin with it, and had obtained better results.

In reply to Dr. Coen's remarks concerning the action of "Salyrgan", Dr. Lidwill said that there was nothing new in the use of mercurial preparations in cardiac œdema. When he had been a student and resident medical officer it had been the custom to give calomel and digitalis, and as much as fifteen grains of *digitalis folia* and thirty grains of calomel had been given in two days; good results had been obtained, the patients passing large quantities of urine. Regarding the action of quinidine, he said that quinidine slowed the auricular rate and was apt to allow impulses to pass through more easily to the ventricle. If, however, large doses of digitalis were given, the beats were checked from passing through.

In reply to Dr. Halliday, Dr. Lidwill said that there was no danger of embolism due to the administration of quinidine unless there was an old-standing auricular fibrillation with clots in the auricles, and in these cases quinidine should never be given.

Dr. Lidwill said that he had been interested to hear Dr. Walker's favourable opinion of "Coramine", as in his experience camphor preparations had not been of much value.

He did not understand the pathological mechanism of pulmonary œdema. *Post mortem* examination of the jockey boy mentioned in his paper had revealed cloudy swelling of the renal tubules and nothing else.

Dr. Lidwill said that Dr. Maddox's suggestion concerning the use of sodium citrate solution in venesection was a good one, but he pointed out that it was seldom, when called to a case of cardiac emergency, that the medical practitioner found such refinements were available. Dr. Lidwill did not agree with the views concerning the advisability of patients moving about after coronary occlusion.

In reply to Dr. Fisher, Dr. Lidwill said that many years previously he had tried out some of the nitrites and had found that if the patient had arteriosclerosis the blood pressure rose far higher than when the blood vessels were normal. The more pronounced the arteriosclerosis, the higher the blood pressure rose. In *angina pectoris* the beneficial action of trinitrin probably consisted in a temporary dilatation of the coronary vessels.

In reply to Dr. McDonald, Dr. Lidwill said that a few years previously Grant had injected the Thebesian vessels and had found that they came to dead ends. The blood was sucked into the Thebesian vessels in diastole and squeezed out of them during systole. Some of the coronary vessels carried blood directly into the chambers of the heart by means of sinuses. In reply to Dr. McDonald's remarks concerning atropine, Dr. Lidwill said that he had tried one one-hundredth of a grain and one-fiftieth of a grain without result; in a few cases only he had given one-thirtieth of a grain and the pulse rate had risen. Atropine poisoning would occasionally develop if such large doses were used. He had a patient who, when given a single morphine and atropine suppository, became incoherent and delirious. Ephedrine could be tried first; if it did not succeed atropine could be used. Dr. Lidwill said that he had obtained some good results with barium chloride, but not so good as those obtained by Dr. Smith; he still used it.

In reply to Dr. McDonald's remarks concerning proprietary medicines, Dr. Lidwill said that he had no interests in Messrs. Burroughs, Wellcome and Company, Limited. He pointed out that the Medical Research Council had verified the work on digitalis carried out by this firm. "Digoxin" had been supplied by Messrs. Burroughs, Wellcome and Company, but the research work had been carried out at University College Hospital, under the guidance of Lewis.

Dr. Smith had asked why Dr. Lidwill used strychnine in pulmonary œdema. Dr. Lidwill replied that he had been "brought up on it"; he had been taught to give strychnine with morphine and atropine, and he continued to do so; he was unable to say why he gave it, but it was probably a continuation of a youthful indiscretion.

NOMINATIONS AND ELECTIONS.

THE undermentioned have been elected members of the New South Wales Branch of the British Medical Association:

Macourt, Alice Margaret, M.B., 1931 (Univ. Sydney), 91, Shirley Road, Wollstonecraft.

Meyers, Edwin Solomon Alexander, M.B., B.S., 1934 (Univ. Sydney), Nimbin.

The Royal Australasian College of Surgeons.

ANNUAL MEETING.

THE seventh annual general meeting of the Royal Australasian College of Surgeons was held in Adelaide from February 28 to March 3, 1934. The meeting was attended by approximately one hundred fellows of the College, including representatives of all the Australian States.

Proceedings began at 8.30 p.m. in the Lady Symon Building, when His Excellency Sir Alex. Hore-Ruthven, V.C., Governor of South Australia, declared the meeting open. This was followed by the formal admission of new fellows, and after this the President of the College, Sir Henry Newland, delivered an address (see page 105).

On Thursday, Friday and Saturday, clinical and operative demonstrations were given at the various Adelaide Hospitals, which was much appreciated by all visiting surgeons.

Election of Fellows.

The following candidates were elected as fellows under the new regulations:

In Surgery.—Victoria: Thomas King, Henry Anthony Phillips, Charles Bernays Melville, Henry Newman Mortensen, Cecil Ashley Marshal Renou. South Australia: John Christian Mayo, Douglas Gordon McKay.

In Ophthalmology.—Victoria: Esmé Vivienne Anderson, William Mitchell Box, William John Lawrence Duncan. South Australia: Geoffrey Howard Barham Black.

In Laryngo-Otology.—Victoria: Arthur Barton Pilgrim Amies, Cecil Nathaniel Love Cantor.

The following candidates were elected as fellows under the old regulations:

Victoria: Charles William Bennett.

Western Australia: Thomas Blake Seed.

Report of Council.

The report of the Council was presented as follows:

Deaths of Fellows.

The Council regrets to report that the following fellows have died since the last annual general meeting of the College: R. Hamilton Russell (Victoria), W. E. Herbert (New Zealand), H. F. Shorney (South Australia).

Council Election.

The ballot for the election of the Council resulted in the reelection of the retiring members as follows: Sir Henry Newland, E. D. Ahern, A. L. Kenny, Balcombe Quick, F. P. Sandes.

Resignation of the Vice-President.

On August 26, 1933, Sir Alexander MacCormick tendered his resignation as Vice-President of the College. In accepting this resignation with regret, a letter of appreciation was sent to Sir Alexander MacCormick, on behalf of the Council and fellows, thanking him for the great services which he had rendered in the past, not only to this College, but to Australasian surgery.

At a meeting of the Council held on August 26, 1933, H. B. Devine was elected as a Vice-President of the College, to fill the vacancy in this position created by the resignation of Sir Alexander MacCormick.

Vacancies Created by the Death of the Censor-in-Chief.

The vacancy in the number of elected members of the Council, created by the death of the Censor-in-Chief, was filled by the appointment of Balcombe Quick for the remainder of the late R. Hamilton Russell's term of office.

On August 26, 1933, Alan Newton resigned his position as Honorary Secretary of the College and was appointed Censor-in-Chief for the remainder of the late R. Hamilton Russell's term of office. At this meeting of the Council the following motion was carried unanimously:

> That an appreciation of the services rendered to this College by Alan Newton be placed on record in the minutes of this meeting of the Council.

Julian Smith, junior, was appointed Honorary Secretary for the remainder of Alan Newton's term of office, and G. R. A. Syme was appointed Honorary Assistant Secretary.

During his term of office Alan Newton conducted the administrative work of the Censor-in-Chief's department. Under the system of admission then in operation it was necessary that a senior surgeon, preferably retired from practice, should act as Censor-in-Chief, as admissions were based, not upon an examination of the candidates, but upon a system of references from other practitioners, which were reported upon by State/Dominion censors and later considered by the Council. The decision of the Council was communicated to the candidates by the Censor-in-Chief.

The introduction of the examination system, conducted by an Australian or a New Zealand Board of Censors, has brought about an alteration in the duties of the Censor-in-Chief, who now becomes, in effect, the Chairman of the Board of Examiners.

The Council therefore considered it desirable, in order to reduce the amount of work which must be done by the Honorary Secretary of the College, to divide the administrative work in the following manner:

(*a*) Duties of the Censor-in-Chief.

1. He shall act as Chairman of the Board of Censors and shall perform all duties laid down under the new regulations governing the admission of fellows. This entails:
   (*a*) The critical examination of work done by all applicants for fellowship.
   (*b*) Correspondence and/or interviews with surgeons who have been teaching applicants for fellowship.
   (*c*) The direction of further work to be done by men whose training has been inadequate at the time of application.
2. He shall conduct negotiations with hospitals and universities in regard to the training of surgeons.
3. He shall attend to the organization and administration of all examinations in which the College is interested.
4. He shall administer research funds under the control of the College.
5. He shall attend to the administration of all teaching activities of the College.

(*b*) Duties of the Honorary Secretary.
The Honorary Secretary shall perform all administrative duties not included in the above categories.

Boards of Censors.

The following fellows were nominated as members of the Board of Censors for the Dominion of New Zealand: Sir Hugh Acland, Sir Louis Barnett, Professor F. Gordon Bell, Sir Donald McGavin, Sir Carrick Robertson, D. S. Wylie; and the following fellows were nominated as members of the Board of Censors for the Commonwealth of Australia: Professor H. R. Dew (New South Wales), L. C. E. Lindon (South Australia), Sir John McKelvey (New South Wales), Balcombe Quick (Victoria), R. B. Wade (New South Wales), B. T. Zwar (Victoria).

The Censor-in-Chief nominated R. V. Hennessy and G. C. Scantlebury to assist the Board in the examination of candidates for fellowship in laryngo-otology, and J. Ringland Anderson and Mark Gardner to assist in the examination of candidates for fellowship in ophthalmology in Melbourne. The following fellows were nominated by the Censor-in-Chief to assist in the examination of a candidate for fellowship in ophthalmology in Adelaide: Brian F. Moore and A. L. Tostevin.

At a meeting held in Melbourne there were ten candidates for fellowship in surgery, three candidates for fellowship in ophthalmology, and four candidates for fellowship in laryngo-otology. The results were as follows:

	Approved.	Rejected.	Deferred.	Total.
General surgery ..	5	5	—	10
Ophthalmology ...	3	—	—	3
Laryngo-otology ..	2	1	1	4
				17

At a meeting held in Adelaide there were four candidates for fellowship in surgery and one candidate for fellowship in ophthalmology. The results were as follows:

	Approved.	Rejected.	Total.
General surgery	2	2	4
Ophthalmology	1	—	1
			5

Total number of candidates 22, of whom 13 were approved.

Primary Fellowship Examination.

The Council is pleased to report that information has been received from the Secretary of the Royal College of Surgeons of England stating it proposes to hold the primary examination for the diploma of fellowship of that College in Melbourne and in Dunedin in November and December, 1934. Professor G. A. Buckmaster and Mr. G. Gordon Taylor will act as examiners in physiology and anatomy respectively. The written paper will be held

simultaneously in Melbourne and Dunedin, beginning on Thursday, November 29, 1934. The *viva voce* examination will begin in Melbourne on Monday, December 3, and in Dunedin on Wednesday, December 12.

Positions of Surgical Assistants at Clinical Schools.

The Council is pleased to report that the creation of training positions for young surgeons as assistants to indoor surgeons has been approved by the following hospitals: Royal Prince Alfred Hospital, Sydney; Royal Alexandra Hospital for Children, Sydney; Adelaide Hospital; Melbourne Hospital; Alfred Hospital, Melbourne; Saint Vincent's Hospital, Melbourne; Children's Hospital, Melbourne; Mater Misericordiæ Hospital, Brisbane.

Post-Graduate Hospital in Melbourne.

Since the last annual general meeting numerous conferences have been held by H. B. Devine and Alan Newton, acting on behalf of the College, with representatives of the staff of the Homœopathic Hospital. It was represented, on behalf of the College, that it was anxious to increase the facilities for post-graduate surgical work in Australia and that it hoped that a plan could be formulated whereby the resources of the Homœopathic Hospital could be made available for this purpose.

The Board of Management and the members of the honorary staff of the Homœopathic Hospital have cooperated with the College in a cordial manner. The Council desires to record its great appreciation of the help given by members of the surgical staff of the hospital in these negotiations, which have now been brought to a satisfactory conclusion.

It has been decided that the name of the hospital will be changed to the "Prince George Hospital" and that the hospital shall give the following courses of post-graduate instruction:

(*a*) Full-time instruction.
(*b*) Courses of lectures and demonstrations.
(*c*) Tutorial classes.

Permanent Headquarters.

In October, 1933, a tender received from J. C. Taylor and Sons Proprietary, Limited, was accepted for the erection of the College building on the old Model School site in Spring Street, Melbourne. Specifications for the building included the erection of the portico in cement and the large doors of the entrance of the building in wood. Mr. Leighton Irwin, the architect of the College, pointed out to the Executive Committee that the appearance of the building would be greatly enhanced if these two structures could be erected in stone and bronze respectively, and he suggested to the Council that every effort should be made to obtain an extra £1,000 to carry out his suggestion. The Council was pleased to report that Mr. F. J. Cato, the father of a fellow of the College, has generously promised to donate £500 towards the extra money required, on condition that another £500 is subscribed for a similar purpose. The building has been in the course of erection for some months and will be completed about July of this year.

At a previous meeting the Council decided that the College building should be opened in March, 1935. The President of the Royal College of Surgeons of England was invited to perform the ceremony declaring the building open at that time, and has accepted. At the last meeting of the Council, consequent upon inquiries from some distinguished overseas surgeons and from the Secretary of the British Medical Association, London, this matter was re-opened and it was decided to change the date of the opening of the College building to September, 1935, in the week preceding the meeting of the British Medical Association in Melbourne. It was felt, however, that before carrying this proposal into effect, the facts of the matter should be placed before the British Medical Association authorities in Melbourne to gain the sanction of that Association to hold the College ceremony the week preceding its meeting. A letter was therefore written to the Secretary of the British Medical Association in Melbourne, placing the facts before him, and this letter was presented to the General Executive Council of the British Medical Association in Melbourne, and the question that the College building should be opened in the week preceding the British Medical Association meeting was approved. The Executive Committee then communicated this suggestion to the Council of the Royal College of Surgeons of England and, from the reply received, it appeared that it would be quite impossible for the President of that College to visit Australia in September, 1935. At the same time the Executive Committee of the College considered the possibility of obtaining the attendance of those surgeons coming out with the British Medical Association delegation at the College meeting. If the opening of the College building were to be held the week preceding that meeting and the visiting surgeons were to attend the College meeting, then it would be necessary for them to leave London at an earlier date than arranged. A cable was therefore sent to the Secretary of the British Medical Association, London, asking if the surgeons visiting Australia at that time might not leave London a week earlier. This was found to be impossible. As the result of these inquiries, the Council of the College decided to adhere to the original proposal to hold the opening ceremony in March, 1935.

Annual General Meeting, 1935.

The next annual general meeting of the College will be held in Melbourne, beginning on Monday, March 4, 1935. The following surgeons have accepted the invitation of the College to attend this meeting: Sir Holburt Waring, Sir D'Arcy Power, Sir William I. de Courcy Wheeler, Professor C. F. Saint, Professor Edward Archibald, D. C. Balfour, Dean Lewis.

The meeting will last for one week and will be devoted almost entirely to surgical work. On Monday afternoon, March 4, the College building will be declared open by the President of the Royal College of Surgeons of England, Sir Holburt Waring. In the evening the inaugural meeting will be held in the Wilson Hall, University of Melbourne, when the third George Adlington Syme Oration will be delivered by Professor F. Wood Jones. In the remaining part of the week operative demonstrations will be held at the Melbourne, Alfred, Saint Vincent's and Children's Hospitals. Surgical papers will be read by surgeons from overseas who are attending the meeting.

Librarian's Report.

The College library is now receiving forty-six journals in exchange for *The Australian and New Zealand Journal of Surgery*. The completed volumes are now in the hands of the binders and will be ready for the library when the College building is opened. At present very few donations of books have been received, but the librarian would be very grateful if intending donors would notify him of any back number journals or surgical literature which they can make available when the College building is completed.

Birthday Honours.

The Council is pleased to report that, since the last annual general meeting, the following fellows have been honoured by His Majesty: Thomas Peel Dunhill, K.C.V.O.; Hugh T. D. Acland, K.B.; F. A. Maguire, C.M.G.

Journal Report.

A report on *The Australian and New Zealand Journal of Surgery* was presented as follows:

Volume II, Numbers 3 and 4, Volume III, Numbers 1 and 2.—Of these issues, two were devoted to the work of clinical schools. January, 1933 (Volume II, Number 3), consisted of articles contributed by the staff of the Melbourne Hospital, and October, 1933 (Volume III, Number 2), of articles from the Royal Prince Alfred Hospital, Sydney.

Financial.

The auditors report that for the year ended January 31, 1933, a loss of £158 6s. was sustained, while for the year ended January 31, 1934, a profit of £96 3s. 11d. was revealed. Both these results were shown after payment by the College to the Journal Section of the *per capita* grant of £1 per fellow *per annum*. The improvement in

the result was effected principally by: (a) a reduction in the printing and publishing expenses, (b) an increase in the revenue received from advertisements.

Negotiations with Butterworth and Company (Australia), Limited, for the Publication of the Journal.

During the year the Executive Committee entered into negotiations with Butterworth and Company (Australia), Limited, medical and law publishers, of London and Australia, with regard to the publication of the journal by them. A definite offer was made to the Executive Committee by Butterworth and Company to publish the journal under a financial arrangement which they set out in this offer. The Executive Committee considered the proposal a very favourable one, and, after consultation with the members of Council, signed an agreement with Butterworth and Company, whereby in future the journal will be published by them. The January, 1934, issue of the journal was the first one published by Butterworth and Company.

Butterworth and Company have retained the services of the editor of the journal, and they have also made arrangements with the Australasian Medical Publishing Company, Limited, to print the journal. The Council has been very satisfied with the manner in which the Australasian Medical Publishing Company, Limited, has printed the journal.

Report of Editorial Committee to the Council.

A report was submitted by the Editorial Committee to the Council as follows:

Volume II, Numbers 3 and 4, Volume III, Numbers 1 and 2.—Of these issues two were devoted to the work of clinical schools. January (Volume II, Number 3) consisted of articles contributed by the staff of the Melbourne Hospital, and October (Volume III, Number 2) of articles from the Royal Prince Alfred Hospital, Sydney. The following committees, appointed for the staffs of the respective hospitals, were responsible for the hospital numbers:

For the Royal Prince Alfred Hospital: Professor Dew, Mr. Douglas Miller, Dr. Corlette, Dr. Susman, Dr. Angel Money.

For the Melbourne Hospital: Mr. B. T. Zwar, Mr. Julian Smith, junior.

As a result of the taking over of the publishing of the journal by Butterworth and Company, the Editorial Committee has, under an agreement, the responsibility of keeping the publisher supplied with suitable scientific matter for the journal. In order to carry on the work of the Editorial Committee during the year, and thus help the publishers, Editorial Executive Committees have been formed as follows:

Sydney: Professor Dew, Mr. Douglas Miller, Dr. Corlette, Dr. Susman, Dr. Angel Money.

Melbourne: Mr. H. B. Devine, Mr. B. T. Zwar, Mr. Balcombe Quick, Mr. Julian Smith, junior.

New Zealand: Sir Louis Barnett, Mr. D. S. Wylie, Professor Gordon Bell.

The Editorial Committee would like to record its appreciation of the manner in which the editor has carried out his duties. It would also like to compliment the Australasian Medical Publishing Company, Limited, on the manner in which it has published the journal.

Correspondence.

WASSERMANN REACTION IN AUSTRALIA.

SIR: A statement in the Beattie-Smith Lectures (THE MEDICAL JOURNAL OF AUSTRALIA, June 9, 1934) calls for correction. In his historical prologue Dr. Latham writes: ". . . he (Dr. Froude Flashman) performed probably the first Bordet-Wassermann tests in Australia." The first man in Australia to experiment with this diagnostic method was Dr. Konrad Hiller, of Melbourne, who read a paper at the Melbourne Congress on the technical procedure and gave an account of experiments by himself in its use for diagnosis.

Yours, etc.,

A. G. BUTLER.

Duntroon,
Federal Capital Territory,
July 9, 1934.

THE TREATMENT OF THE FAILING HEART.

SIR: On glancing through the recent article on "The Treatment of the Failing Heart", I was amazed, in this year of grace, to read (page 846): ". . . oxygen, best given by nasal catheter passed into the pharynx . . . a just continuous stream of bubbles is maintained." Seven years ago Gilchrist and I made a careful quantitative assessment of the efficiency of various methods of oxygen administration (*Edinburgh Medical Journal*, May, 1925) and found the method above described to be hopelessly inadequate and inefficient. It has no advantages over the glass funnel method, which has now been almost universally abandoned. From personal experience I can state that the nasal catheter is at best uncomfortable and, with an effective rate of oxygen flow (not less than three to four litres per minute for an adult), intolerable. Those who have used oxygen efficiently are, I think, agreed that it is a valuable life-saving remedy. But the use of inadequate methods and the withholding of oxygen until the patient is moribund have combined to bring this valuable remedy into disrepute.

In the subsequent paragraph a prescription is given which combines morphine, heroin and apomorphine. As the two former are extremely selective and potent depressors of the cough reflex, while the latter is a powerful nauseant expectorant, surely this is an example of therapeutic incompatibility.

Yours, etc.,

H. WHITRIDGE DAVIES.

Department of Physiology,
The University of Sydney,
July 10, 1934.

LOW BACKACHE.

SIR: Your very positive correspondent, Dr. Stewart McKay, who, in the issue of June 30, displays so much talent for humour at other people's expense, might like a further opportunity of amusing us.

I am sure that, though not as brilliant as a gathering of Mayo surgeons, we may be able still to penetrate the subtlety of his wit.

I would therefore ask: Is "tiredness" a symptom, a psychological mood or a metabolic state?

Yours, etc.,

W. McRAE RUSSELL, M.B., B.S.

Camberwell,
Melbourne,
July 10, 1934.

Obituary.

GEORGE THOMAS HOWARD.

WE regret to announce the death of Dr. George Thomas Howard, which occurred on July 17, 1934, at Hawthorn, Victoria.

Books Received.

ALCOHOL AND ANÆSTHESIA, by W. Burridge, D.M., M.A.; 1934. London: Williams and Norgate. Demy 8vo., pp. 65. Price: 2s. 6d. net.

CLIO MEDICA: A SERIES OF PRIMERS ON THE HISTORY OF MEDICINE, edited by E. B. Krumbhaar, M.D.; XII: Japanese Medicine, by Y. Fujikawa, M.D., translated by J. Ruhräh, M.D.; 1934. New York: Paul B. Hoeber. Foolscap 8vo., pp. 127. Price: $2.50 net.

POCKET MONOGRAPHS ON PRACTICAL MEDICINE: BLOOD DISEASES IN GENERAL PRACTICE, by A. Piney, M.D., M.R.C.P.; 1934. London: John Bale, Sons and Danielsson. Foolscap 8vo., pp. 92. Price: 2s. 6d. net.

ULTRA-VIOLET THERAPY IN EYE DISEASE, WITH A REVIEW OF THE ACTION OF OTHER FORMS OF RADIANT ENERGY, by F. W. Law, M.A., M.D., B.Chir., F.R.C.S., with a foreword by S. Duke-Elder, M.A., D.Sc., M.D., Ph.D., F.R.C.S.; 1934. London: John Murray. Demy 8vo., pp. 88. Price: 5s. net.

Diary for the Month.

AUG. 1.—Western Australian Branch, B.M.A.: Council.
AUG. 2.—South Australian Branch, B.M.A.: Council.
AUG. 2.—Victorian Branch, B.M.A.: Clinical Meeting.
AUG. 3.—Queensland Branch, B.M.A.: Branch.
AUG. 6.—New South Wales Branch, B.M.A.: Organization and Science Committee.
AUG. 7.—Tasmanian Branch, B.M.A.: Council.
AUG. 8.—Victorian Branch, B.M.A.: Branch.
AUG. 10.—Queensland Branch, B.M.A.: Council.
AUG. 11.—New South Wales Branch, B.M.A.: Executive and Finance Committee.
AUG. 14.—Tasmanian Branch, B.M.A.: Branch.
AUG. 15.—Western Australian Branch, B.M.A.: Branch.
AUG. 15.—Victorian Branch, B.M.A.: Clinical Meeting.
AUG. 21.—New South Wales Branch, B.M.A.: Ethics Committee.
AUG. 21.—Tasmanian Branch, B.M.A.: Council.
AUG. 22.—Victorian Branch, B.M.A.: Council.
AUG. 23.—New South Wales Branch, B.M.A.: Clinical Meeting.
AUG. 24.—Queensland Branch, B.M.A.: Council.
AUG. 28.—New South Wales Branch, B.M.A.: Medical Politics Committee.

Medical Appointments.

Dr. J. G. Cameron (B.M.A.) has been appointed, pursuant to the provisions of the *Workers' Compensation Act*, 1928, Certifying Medical Practitioner at Heidelberg, Victoria.

* * *

Dr. H. K. Fry (B.M.A.) has been reappointed an Official Visitor to the Parkside Mental Hospital, South Australia.

* * *

Dr. L. M. L. Hungerford has been appointed Medical Officer of Health by the Esperance Road Board, Western Australia.

* * *

Dr. B. L. Harbison (B.M.A.) has been appointed Government Medical Officer at Emerald, Queensland.

* * *

Dr. H. W. Harbison has been appointed Government Medical Officer at Blackall, Queensland.

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Medical Appointments Vacant, etc.

For announcements of medical appointments vacant, assistants, *locum tenentes* sought, etc., see "Advertiser," pages xvi, xvii.

BRITISH MEDICAL ASSOCIATION, VICTORIAN BRANCH: Medical Secretary.

CHILDREN'S HOSPITAL (INCORPORATED), PERTH, WESTERN AUSTRALIA: Junior Resident Medical Officers.

LAUNCESTON PUBLIC HOSPITAL, LAUNCESTON, TASMANIA: Resident Medical Officer (male).

MATER MISERICORDIÆ CHILDREN'S HOSPITAL, BRISBANE, QUEENSLAND: Resident Medical Officer.

MILDURA DISTRICT HOSPITAL, MILDURA, VICTORIA: Junior Resident Medical Officer.

PERTH HOSPITAL, PERTH, WESTERN AUSTRALIA: Resident Medical Officers.

Medical Appointments: Important Notice.

MEDICAL practitioners are requested not to apply for any appointment referred to in the following table without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, Tavistock Square London, W.C.1.

BRANCH.	APPOINTMENTS.
NEW SOUTH WALES: Honorary Secretary, 135, Macquarie Street, Sydney.	Australian Natives' Association. Ashfield and District United Friendly Societies' Dispensary. Balmain United Friendly Societies' Dispensary. Friendly Society Lodges at Casino. Leichhardt and Petersham United Friendly Societies' Dispensary. Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney. North Sydney Friendly Societies' Dispensary Limited. People's Prudential Assurance Company Limited. Phœnix Mutual Provident Society.
VICTORIAN: Honorary Secretary, Medical Society Hall, East Melbourne.	All Institutes or Medical Dispensaries. Australian Prudential Association, Proprietary, Limited. Mutual National Provident Club. National Provident Association. Hospital or other appointments outside Victoria.
QUEENSLAND: Honorary Secretary, B.M.A. Building, Adelaide Street, Brisbane.	Brisbane Associated Friendly Societies' Medical Institute. Chillagoe Hospital. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL are advised, in their own interests, to submit a copy of their agreement to the Council before signing. Lower Burdekin District Hospital, Ayr.
SOUTH AUSTRALIAN: Secretary, 207, North Terrace, Adelaide.	Combined Friendly Societies, Clarendon and Kangarilla districts. Officer of Health, District Council of Elliston. All Lodge Appointments in South Australia. All Contract Practice Appointments in South Australia.
WESTERN AUSTRALIAN: Honorary Secretary, 205, Saint George's Terrace, Perth.	All Contract Practice Appointments in Western Australia.
NEW ZEALAND (Wellington Division): Honorary Secretary, Wellington.	Friendly Society Lodges, Wellington, New Zealand.

Editorial Notices.

MANUSCRIPTS forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary be stated.

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Members and subscribers are requested to notify the Manager, THE MEDICAL JOURNAL OF AUSTRALIA, Seamer Street, Glebe, New South Wales, without delay, of any irregularity in the delivery of this journal. The management cannot accept any responsibility or recognize any claim arising out of non-receipt of journals unless such a notification is received within one month.

SUBSCRIPTION RATES.—Medical students and others not receiving THE MEDICAL JOURNAL OF AUSTRALIA in virtue of membership of the Branches of the British Medical Association in the Commonwealth can become subscribers to the journal by applying to the Manager or through the usual agents and booksellers. Subscriptions can commence at the beginning of any quarter and are renewable on December 31. The rates are £2 for Australia and £2 5s. abroad *per annum* payable in advance.